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BY

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And in Keen Appreciation of

HIS WIDE-SPREAD SYMPATHIES AND GENEROUS IMPULSES,

This Work is Dedicated

BY

THE AUTHOR.

PREFACE TO THE SECOND EDITION.

SINCE the appearance of the First Edition of this Treatise great advances have been made in our knowledge of the intimate structure of the Nervous System. The new methods of Golgi and his School have thrown light on many obscure points in neuro-histology, and the doctrine of the neuron and neuron chains has had a paramount influence upon problems in nerve physiology and pathology.

It is hoped that the revision of the Anatomical Section faithfully reflects the change which has come over our conception of the nerve-cell, and that this section correctly and concisely expresses the more essential facts which recent research has established. The large number of additional plates and illustrations in the text will, it is believed, be welcomed in this section: most of them are from original drawings by the Author.

In the Clinical Section will be found, as additional matter, a chapter on Progressive Systematised Insanity; an account of certain forms of Impulsive Insanity; a description of the reaction-time instrument; estimation of muscular sense discrimination in general paralysis, alcoholism, &c.; and a separate chapter on the Treatment of Insanity. Much matter considered less essential has been eliminated.

In the Pathological Section, the Author has maintained his views of the importance of the Scavenger-cell as a morbid factor in the changes found in the brain of the insane, nor does he find in recent criticism of this view any solid reason for a modification of his opinion as to the rôle played by it in insanity.

Finally, he would express his grateful appreciation of the flattering reception accorded to the First Edition, and his great indebtedness to his Publishers, who have spared neither expense nor care in the production of the Second Edition.

WEST RIDING ASYLUM,
WAKEFIELD, *December*, 1898.

PREFACE.

In writing a new Treatise on Mental Diseases, I have not been blind to the wealth of available literature in this department, nor to the claims upon the Student's attention of such works as the classical Manual of Bucknill & Tuke, the English translation of Griesinger's Treatise, and the admirable Lectures of Dr. Blandford, nor again to the more recent additions to Dr. Bristowe's *Clinical Medicine,* and the works of Drs. Sankey, Clouston, and Savage.

It has, however, been my special object to present a *résumé* of our knowledge of the structure and connections of the cerebro-spinal nervous system, of the architecture of the cerebral hemispheres, and more especially of the cortical envelope as the essential organ—the material substratum—of Mind; and to afford a concise account of the morbid changes found in the brain of the insane, as viewed in the light of recent research.

It appears to me that a disproportionate amount of attention has been paid in former text-books to the clinical aspects of Insanity, and it is hoped that this attempt to deal more fully with the organisation of the material substratum of mind, and with the evidences of morbid change to which it is prone, will not prove unwelcome to the Student of Mental Disease.

In the *Anatomical Section,* I have endeavoured to comprise such information as shall prove of utility to a more thorough conception of the ground-plan and superstructure of the nervous system; and it will be at once evident that special emphasis has been advisedly bestowed upon the cortical envelope—the structure, nature, and autonomy of the nerve-cell.

The *Clinical Section* comprises statistics based upon an analysis of 4,000 cases of Insanity in both sexes, treated at the West Riding Asylum.

In the *Pathological Section,* I have endeavoured to do justice to certain morbid processes, which appear to me to be of paramount importance in the history of Insanity; and more particularly would I here allude to the functions of the Lymph-connective system of the Brain, and the life-history of the "Scavenger-cell."

To my Publishers I would desire to express my acknowledgments for the consideration uniformly received at their hands, despite the delay which has unavoidably occurred—for the liberal supply of illustrations, so essential to the success of a work of this description, and for the special care taken in their production. I can add my testimony to the admirable faithfulness with which my drawings have been reproduced by Mr. Danielsson.

I have also to express my obligations to my colleague, Mr. St. John Bullen, for reference to a compilation of Statistics from the Pathological Records of this Asylum, and for material assistance in the revision of the proof-sheets.

West Riding Asylum,
Wakefield, *November*, 1889.

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DESCRIPTION OF PLATES.

PLATE I.

ILLUSTRATIVE OF THE "MOTOR TYPE" OF CORTEX. Page 62.

The section taken from the extra-limbic area of rabbit's brain, near the frontal pole of hemisphere. × 200.

The three small figures represent the mesial, basal, and coronal aspect respectively of the rabbit's brain, showing distribution of the various types of cortex.

A. Sub-frontal and sub-parietal segments of the limbic fissure. B. Gyrus hippocampi or lower limbic arc. C. Limbic fissure. D. Occipital pole. E. Frontal pole. F. Olfactory bulbs. G. Optic tract. I. Olfactory root. J. Corpus callosum. K. Parietal sulcus. S. Sylvian depression. T. Limbic fissure. The types of cortex are indicated by the subjacent scheme.

PLATE II. Page 70.

FIG. 1. CEREBELLAR CORTEX OF YOUNG RAT.

Section in direction of lamina, showing the arrangement of cells of Purkinje, and the T-shaped fibres arising from the underlying granules. Sublimate preparation. × 110.

FIG. 2.

Section across lamina showing arborisation of dendrites of cells of Purkinje and their descending axons. Sublimate preparation. × 110.

PLATE III. Page 74.

FIG. 1. CEREBELLAR CORTEX OF MOUSE.

Showing cells of Purkinje, granules, and moss-fibres. Sublimate preparation. × 110.

FIG. 2. CEREBELLAR CORTEX OF MAN.

Showing stellate cells in neighbourhood of cells of Purkinje, distributing branches upwards to the peripheral zone; section taken across lamina. Sublimate preparation. × 110.

PLATE IV. Page 82.

FIG. 1. CEREBELLAR CORTEX (HUMAN).

Section in direction of lamina shows large stellate nerve-cell near cells of Purkinje; the latter are truncated; a few terminal dendrites occupy outer half of peripheral zone, the horizontal fibres being derived from the granules below. Sublimate preparation. × 110.

FIG. 2. OLFACTORY BULB OF RAT (SEMI-DIAGRAM).

Arrangement of mitre-cells in relationship to their centric and peripheric distribution.

PLATE V. Page 92.

FIG. 1. ILLUSTRATIVE OF THE "MOTOR TYPE" OF CORTEX.

Taken from the left hemisphere of the brain of the pig to exhibit its five-laminated type with the nests or clustered ganglionic cells. × 76.

FIG. 2. "MOTOR CORTEX" OF PIG.

Nerve-elements of second, third, and fourth layers respectively. × 306.

PLATE VI. Page 100.

FIG. 1. ILLUSTRATIVE OF THE "SENSORY TYPE" OF CORTEX.

Taken from the first annectant gyrus of human brain. × 65.

FIG 2. "SENSORY TYPE" OF CORTEX.

Nerve-elements of the third, fourth, fifth, and sixth layers of first annectant gyrus of human brain. × 157.

PLATE VII. Page 104.

FIG. 1. CEREBRAL CORTEX: NERVE-CELLS OF SECOND LAYER (RAT).

Showing characteristic groupings of cells: the dendrites are notably hirsute, and the terminal dendrites of a large nerve-cell from the subjacent layers pass upwards betwixt the two groups. Sublimate preparation. × 110.

FIG 2. CORNU AMMONIS OF YOUNG RAT.

Shows pyramidal nerve-cells of Cornu and granules of fascia dentata. Sublimate preparation. × 110.

PLATE VIII. Page 108.

FIG. 1. CEREBELLAR CORTEX OF MOUSE.

Shows granular layer between cells of Purkinje, and the T-shaped fibres of peripheral zone. Sublimate preparation. × 110.

FIG. 2. CEREBRAL CORTEX OF RAT.

Arrangement of stellate connective element beneath pia covering peripheral zone; transitional elements are also shown interblending with the terminal arborisations from nerve-cells of underlying strata. Silver-chrome preparation. × 110.

PLATE IX. Page 114.

FIG. 1. CEREBELLAR CORTEX (MOUSE).

Shows basket-work around cells of Purkinje: axons from intrinsic cells of peripheral zone are seen contributing by numerous descending collaterals to this basket-work, which in the specimen is much obscured by the dense deposit of sublimate. Sublimate preparation. × 360.

FIG. 2. SCHEME OF RETINAL LAYERS IN MAMMALS.

As indicated by the more recent researches of Ramon-y-Cajal.

PLATE X. Page 116.

FIG. 1. "SENSORY CORTEX."

Taken from the "modified upper limbic" type in the brain of the rabbit. This area is represented in the small figures on the rabbit's brain on this plate by the dotted area covering the inner and mesial aspect posteriorly, internal to the parietal sulcus, K.

FIG. 2. "MODIFIED OLFACTORY TYPE."

Taken from the posterior extremity of the lower limbic arc of the rabbit's brain. The area is represented in the first of the three figures, and is lettered T. The large swollen cell of the second layer is a notable feature of this cortical area. × 210.

PLATE XI. Page 124.

FIG. 1. CEREBRAL CORTEX (RAT).

Arrangement of nerve-cells of cortex beneath "outer olfactory root." Silver-chrome preparation. × 110.

FIG. 2. CEREBRAL CORTEX OF RAT.

Nerve-cells showing extremely hirsute dendrites and descending axons. Sublimate preparation. × 110.

PLATE XII. Page 132.

SECTION THROUGH "MOTOR CORTEX" OF BRAIN OF CAT.

Specially prepared to show relationships existing between the nerve-cells and the lymphatic channels and saccules of cortex. The connection of the pericellular sacs with the blood-vessels is clearly indicated, as is also the arching of the nutrient vessel around the nerve-cell. The perivascular nuclei in some cases alone indicate the position of the lymphatic sheath.

PLATE XIII. Page 136.

FIG. 1. SPINAL GANGLION-CELL OF THE FROG (VON LENHOSSEK).

The nucleus shows a deep stained nucleolus, a faint linin network beset with chromatin granules. The filar element is faintly represented in the cytoplasm, but the chromophilic granules of Nissl are a notable feature, together with the finer granular vortex beneath the nucleus, representing the so-called centrosome. Two small connective tissue cells appear to the left. This figure is borrowed from Wilson's *The Cell in Development and Inheritance*.

FIG. 2. CORTEX OF CEREBRUM (PIG, TWO DAYS OLD).

Sublimate preparation taken from near mesial aspect of hemisphere. Sublimate preparation. × 110.

PLATE XIV. Page 512.

FIG. 1. DEGENERATION OF MEDULLATED FIBRES IN LATERAL COLUMNS OF SPINAL CORD FORMING SO-CALLED "MILIARY SCLEROSIS," AS SEEN UNDER A LOW-POWER OBJECTIVE.

The pale nodular structures are the "miliary" patches into which varicose and moniliform fibres are seen to pass; most of such patches are multilocular, and are surrounded by deep-stained sclerosed tissue.

FIG. 2. "COLLOID" PATCHES RESULTING FROM DEGENERATION OF MEDULLATED FIBRES OF SPINAL CORD—MORE HIGHLY MAGNIFIED.

The multilocular constitution of the patch is indicated by delicate outlines; axis-cylinders devoid of medulla are seen passing into the degenerated focus; and scavenger-cell is seen thrusting its ramifying processes into the substance of the colloid patch.

FIG. 3. "COLLOID" PATCH STILL MORE HIGHLY MAGNIFIED TO SHOW THE OUTLINE OF MULTILOCULAR MATERIAL WITH A FINE STROMA OF ELASTIC FIBRES AROUND. × 350.

PLATE XV. Page 516.

DEGENERATION OF NERVE-FIBRES OF LATERAL COLUMNS OF SPINAL CORD IN SO-CALLED "COLLOID DEGENERATION" OF THESE TRACTS.

The axis-cylinders are seen stripped of their medullated sheath, or surrounded by moniliform medulla undergoing granular degeneration, each with a superimposed nucleus, which really represents a young scavenger-cell. At other parts of the field the medulla is seen in process of segmentation, and attacked by scavenger-cells crowded with granular contents.

PLATE XVI. Page 520.

"COLLOID DEGENERATION."

Portion of inferior olivary and accessory olivary bodies in a case of glosso-labio-laryngeal paralysis (*T. W.*, p. 520), showing spheroidal products of degenerated medullated fibre, and the complete immunity from morbid change presented by the grey matter. × 22.

PLATE XVII. Page 526.

FIG. 1. "COLLOID DEGENERATION."

Showing degeneration of the medullated arciform fibres of the first layer of the cortex in a case of chronic alcoholic insanity. A deep-stained sclerous belt bounds the outer zone, crowded with degenerated products of medullated fibre forming "colloid" bodies arranged in linear series. Active scavenger-cells are seen scattered profusely throughout the morbid patch. × 350.

FIG. 2. "COLLOID" TRANSFORMATION OF MEDULLATED FIBRE FORMING THE ARCIFORM STRIPE OF THE PERIPHERAL ZONE OF THE CORTEX IN A CASE OF ADVANCED SENILE ATROPHY OF THE BRAIN.

Numerous scavenger-cells are scattered amongst the degenerate fibres, and oil-globules crowd upon the vessels in their vicinity. × 350.

PLATE XVIII. Page 536.

TO ILLUSTRATE SCLEROSIS OF CEREBELLAR CORTEX IN A CASE OF EPILEPTIC IMBECILITY.

A. Healthy leaflet closely adjoining diseased tract, showing the cells of Purkinje uninvolved.

B. The sclerosed leaflets united firmly together, the cells of Purkinje absent, and the normal structure completely altered.

PLATE XIX. Page 544.

TO ILLUSTRATE SCLEROSIS OF CORNU AMMONIS IN EPILEPTIC INSANITY.

A. Peripheral zone in gyrus hippocampi.

B. Vacuolated cells beneath the above.

PLATE XX. Page 552.

VACUOLATION OF NUCLEI OF NERVE-CELLS.

To illustrate extreme degrees of this change in the elements of the second and third layers of the cortex—human brain. The nucleus, which in the normal state should stain much deeper than the cell, remains colourless or is swollen into one large vacuole. At times such a vacuole appears to have burst through the cell-protoplasm, or the latter contains numerous small vacuoles, the unaffected protoplasm still staining deeply.

PLATE XXI. Page 556.

EXTREME DEGREES OF VACUOLATION IN THE MULTIPOLAR GANGLIONIC CELLS OF SPINAL CORD.

The coincidence of granular degeneration, and vacuolation is here seen. The cells are swollen with bright translucent contents, or indurated and devoid of branches. The retracted protoplasm and displaced nucleus are evident features. × 350.

PLATE XXII. Page 560.

FUSCOUS DEGENERATION OF LARGE GANGLIONIC CELLS FROM THE MOTOR CORTEX OF HUMAN BRAIN.

The pale patches represent the areas of pigmentary degeneration, the mass being coarsely granular and of bright yellow tint. The unaffected protoplasm is seen retracted and stained by the aniline dye, and the apex of one of these cells is pigmented and stunted. × 350.

PLATE XXIII. Page 568.

FIG. 1. FATTY DEGENERATION AND ATROPHIC SHRINKING OF THE PERIPHERAL ZONE (1ST LAYER) OF CORTEX IN A CASE OF ADVANCED SENILE ATROPHY OF THE BRAIN, TAKEN FROM A SECTION OF THE ASCENDING FRONTAL CONVOLUTION.

Scavenger-elements are seen profusely scattered through the upper layers, invading the vascular-tracts and surrounding the nerve-cells. The latter are diminished in numbers, and a notable sclerous shrinking of these superficial layers of the cortex is evident. × 130.

FIG. 2. FATTY DISINTEGRATION OF CORTEX IN A CASE OF ADVANCED SENILE ATROPHY OF THE BRAIN.

The peripheral zone (first layer) is here delineated, the vascular walls as well as the scavenger-cells are surrounded by collections of oil-globules and fatty débris. × 350.

FIG. 3. NERVE-CELLS FROM THE DEEPER LAYERS OF THE CORTEX UNDERGOING DISINTEGRATION AND REMOVAL BY THE AGENCY OF SCAVENGER-CELLS WHICH SURROUND THEM.

The nerve-cells are seen in different stages of degeneration—swollen, irregular, and deformed, devoid of branching processes, or reduced to a formless heap of granular débris still enclosing the nucleus. The scavenger-cells contain numerous coarse granules in their interior, deeply stained and similar to the products of nerve-disintegration around them. × 350.

PLATE XXIV. Page 584.

FIG. 1. GRANULAR DEGENERATION OF NERVE-CELLS.

Taken from the fifth layer of motor cortex in a case of chronic alcoholic insanity. Proliferation of the perivascular nuclei as well as the pericellular elements is evident, and a coarse granular degeneration of the protoplasm of the nerve-cell has occurred. × 210.

FIG. 2. INVASION OF DEEPEST OR SPINDLE-CELL LAYER OF THE CORTEX BY SCAVENGER-CELLS IN A CASE OF CHRONIC ALCOHOLIC INSANITY.

The blood-vessels which are undergoing fatty degeneration are crowded by perivascular nuclei and surrounded by numerous scavenger-elements. × 180.

FIG. 3. ANEURYSMAL DILATATION OF PERIVASCULAR SAC IN A CASE OF GENERAL PARALYSIS. × 210.

PLATE XXV. Page 588.

SCAVENGER-ELEMENTS IN FIRST LAYER OF CORTEX OF HUMAN BRAIN, ILLUSTRATING THEIR MODE OF PROLIFERATION AND PERMEATION OF THE CORTICAL STRUCTURE BY THEIR FIBRILLATION.

The coarse vascular processes may readily be distinguished from the finer fibrils given off by these organisms. × 240.

PLATE XXVI. Page 592.

DEGENERATION OF NERVE-CELLS IN HUMAN CORTEX.

Taken from a section of the motor region to illustrate the mode of connection and relationships existing between the scavenger-elements of the lymph-connective system and the cortical blood-vessels. The swollen granular degenerated nerve-cells are seen attacked on all hands by the scavenger-elements. × 210.

DESCRIPTION OF CHARTS.

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CHART B. Chart of recoveries in insanity of puberty and adolescence in both sexes. Page 396.
CHART C. Chart of recoveries in insanity of the puerperal and lactational periods. Page 422.

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A TREATISE
ON
MENTAL DISEASES.

PART I.—ANATOMICAL AND HISTOLOGICAL SECTION.

Contents.—The Spinal Cord—The Medulla Oblongata—The Mesencephalon—The Thalamencephalon—The Prosencephalon—The Encephalon as a whole—The Cerebral Cortex—Cortical Lamination.

THE SPINAL CORD.

The cerebro-spinal axis consists of a series of longitudinally disposed columns of white medullated nerve fibre, arranged around a central axis of grey ganglionic substance, which in its turn surrounds a central cavity or cavities. Both white and grey columns constitute symmetrical and bilaterally disposed halves, reminding us of the double ganglionic cord in the *invertebrata*, and are connected across the middle line by a system of commissural fibres, and by certain tracts of the white columns which decussate from the one half into the other at different levels of the system. We have certain points to allude to, both as regards the idea of "medullated columns" and the central "grey axis," as well as the "bilateral symmetry" referred to, ere we describe in detail the structures themselves.

In the first place, the central grey axis surrounding the central cavity should not be considered altogether in the light of a uniform column of grey matter, but rather as a series of ganglionic masses, which, fused together along the whole length of the spinal cord, become dissevered into separate masses in the **medulla oblongata,** and into the much larger and more important ganglionic masses at the base of the **cerebrum.** Although fused in the spinal cord, an indication of the primitive ganglionic type of the *invertebrata* can still be traced in the enlargement of the grey substance at the level of each spinal nerve, mapping off, as it were, each spinal segment from its neighbour above and below it.

In the next place, the columns of white medullated fibre are not *continuous tracts* throughout the whole cerebro-spinal axis; they constitute rather a multiplicity of smaller columns, each of which varies in its destination, and consequently in its longitudinal extent. We may correctly presume that—taking as the *longest course* pursued by these medullated tracts that of fibres arising from the grey cortex of the cerebrum, and passing down the whole length of the cord to terminate in the motor cells for the lower extremities in the lumbar region—we have between these and the shortest every intermediate length of medullated fibre, interrupted by the ganglionic masses to which it is destined. The *shortest* fibres will probably be a series of fibres running as *longitudinal commissures* between the neighbouring spinal segments alluded to: these occur in the anterior and posterior columns of the cord.

We have spoken of the fibres as being "interrupted" by the grey matter, by which we must understand the important fact, that at such points a functional connection is established between the nerve fibres and the nerve cells which abound in the grey matter at these *points of interruption*, and from which cells a fresh start of fibres is made into other realms.

With respect to the bilateral symmetry of these two halves of the cerebro-spinal axis, it must be also stated that although at a first glance the various parts constituting the brain, medulla, and spinal cord, would appear to exactly reproduce such symmetry of arrangement, yet in the former, a lateral asymmetry is detected by a more careful consideration of its cortical envelope, the convolutionary surface of which varies very considerably in either hemisphere as regards *arrangement* and *complexity* of gyri, and *superficial area* of grey matter. This bilateral asymmetry apparently conforms to the extreme differentiation in structure, accompanying the more independent functional activity of the cerebral hemispheres, and histological research teaches us still more forcibly how infinite become the possibilities for this hemispheric differentiation.

We must likewise attend to the reversal of conditions in the case of the white and grey matter constituting the large cranial ganglionic structures and the spinal cord respectively. In the latter—the spinal cord—the grey matter is central, and is invested *externally* by the white nerve fibre; in the former, as the **cerebral hemispheres** and **cerebellum,** the white medulla is central, and invested externally by an envelope of grey cortex. We need only state here that the last is the type assumed by those ganglionic levels which form the starting-point of fibres for centric destinations: the first is the type assumed for the reception of such *centric diffusions.* Wherever centric fibres terminate in this radiate manner, there the grey cortex assumes a sort of outer capsular investment and the medullated fasciculi its centric core. We shall find this appertain to the radiating fibres received by the **cerebrum,** the **cerebellum,** the **thalamus, quadrigeminal bodies,** the **geniculate,** and the **inferior olivary,** in particular; and, in almost all alike, we shall find even to the cerebral hemispheres, a zonular layer of medullated fibres bounding the grey capsular investment.

To revert to the lower or subordinate levels, or the *spinal* axis, we learn to familiarise ourselves with the disposition and longitudinal direction of its various columns and ganglionic centres, by studying a series of sections taken in different planes and at various levels. For our present purpose, however, it is but necessary to acquaint ourselves with the appearance of its parts as seen in *transverse* section. In such a section carried through the *lumbar* enlargement of the cord, we see the irregularly crescentic masses of grey matter disposed on either side and connected across the median line by the **anterior and posterior commissures,** between which lies the minute orifice of the central canal. The anterior cornu or horn is at this site thick, broad, and bulbous; the posterior horn, as in other regions, is longer and narrower, directed towards the groove on the outer surface of the cord, which separates the lateral from the posterior columns, and where it receives the lateral section of the fibres of the posterior roots. Somewhat expanded at its extremity, the posterior cornu is obliquely truncated from within outwards, and capped at this site by a translucent substance, the **substantia gelatinosa of Rolando;** the expanded part so capped, and forming the greater part of the posterior horn, being called the **caput.** The connection between it and the anterior horn and median grey is called the **cervix** or **neck** of the horn. Whilst the lateral segment of the posterior roots passes into the **caput cornu,** the median-lying fasciculi arch inwards around the gelatinous substance and ascend in the outermost zones of the posterior column to enter the horn at a higher level, whilst others bend downwards into the grey tract. Just anterior to these arched fibres, where they enter the *cornu,* and on either inner side of the neck of the horn, is found in the upper lumbar region an insignificant cluster of nerve cells, which at higher levels become an important feature, the **vesicular column** of Lockhart Clarke.

In transverse sections the cells of this column look inflated and spherical; they are really fusiform, as seen in longitudinal sections.* Commencing above the *third* lumbar nerve, this formation extends up to the *ninth* dorsal, and in still higher regions are found, occasionally distinctly clustered, similar cells which appear as the representatives of the same formation. Dr. Ross traces this formation as reappearing in the lower end of the medulla oblongata, where he considers it to be represented by the nucleus common to the origin of the spinal accessory, vagus, and glosso-pharyngeal nerves.

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* These cells have been spoken of as "bipolar," but this is an error, since few of the elements fail to show several processes.

A similar formation appears in the sacral region (origin of the second and third sacral nerves) as the **sacral nucleus of Stilling**; so that we have throughout the length of the spinal cord and lower end of medulla an interrupted column appearing at the two extreme ends and in the thoracic division, of which the latter is far the more conspicuous, and lies exactly along the plane of emergence of the visceral nerves (*Gaskell*).

In the posterior horn, we find sparsely scattered cells of fusiform contour and of small size (15μ), which are regarded as sensory elements probably in connection with the posterior roots.

The anterior horn presents in cervical and lumbar regions most conspicuous groups of multipolar cells, which vary much in their distribution with changes in the form of the grey matter; these groupings are fewer and far less conspicuous in the narrow anterior cornu of the dorsal region. The more important groups to be distinguished are five—viz., an inner or median, an anterior, an antero-lateral, a postero-lateral, and a central.

Of these clusters the first and last (median and central) are the least constant; and, in the lumbar region in particular, do we note the absence of the inner or median groups, although even in this region *minute elements* tend to appear occasionally in a somewhat clustered arrangement along the mesial border of the grey matter; at all times the inner is one of the least prominent clusters in the horn.

Of the antero-lateral and postero-lateral groups, the latter is the more conspicuous, both as regards size of cluster, dimensions, and number of cells.

These two groups occupy the outer margin of the grey horn, the one lying in front of the other, and usually occasioning a well-defined anterior and posterior angular projection of the horn.

Internal to these, between them and the inner, and behind the anterior, lies in certain regions a central cluster, also a well-defined group.

In the upper dorsal and lower cervical region a prominent lateral projection from the outer side of the grey matter betwixt anterior and posterior horn has long been known as the **intermedio-lateral tract** of Clarke: it contains a cluster of cells which higher up fuse with the postero-lateral group already alluded to.

The outer margin of grey is behind this site blended in a coarse meshwork with the neighbouring white medullated strands, constituting the so-called **formatio reticularis.**

The white medullated substance of the cord investing this central grey mass is roughly distinguished into an anterior, lateral, and posterior column—the former extending to the outermost roots of the motor nerves: the second from this point back to the attachment of the sensory roots: and the last to the posterior median fissure. These columns are each of them further mapped out into separate tracts, indicated *anatomically* by a distinct groove on the surface and by a difference in the dimension of their fibres; or by the results of *embryological* research indicating their *medullated* development at distinct periods of life; or again, by the facts of the Wallerian degeneration resulting from physiological experimentation or the processes of disease.

By one or other of these means we ascertain that at least eight *physiological tracts* may be distinguished in the white substance of the spinal cord. These may be classed as follows:—

Anteriorly—1. Anterior or direct pyramidal tract (also termed the *column of Türck*).
2.—Anterior radicular (or root) zone (also termed *anterior ground fibres of Flechsig*).
Laterally— 3. Lateral or crossed pyramidal tract.
4. Direct cerebellar tract.
5. Antero-lateral ascending tract of Gowers (also extending forwards).
6. Lateral limiting layer.
Posteriorly—7. Postero-external (or postero-lateral) column (*column of Burdach: posterior radicular zone: posterior ground fibres of Flechsig*).
8. Postero-internal (or postero-median) column (*column of Goll*).

Direct Pyramidal Tracts.—A certain proportion of the fibres of the anterior pyramids which escape decussation in the medulla, descend direct on the same side of the cord, forming the tract which more or less completely bounds the anterior median fissure. This anterior pyramidal tract progressively diminishes in size from above downwards, as it becomes distributed to the anterior cornu of the *opposite* side by a continuous decussation of its fibres throughout its course along the anterior commissure. It usually extends to the mid-dorsal region, but occasionally passes down as far as the lumbar cord, and is found to vary very considerably in size for the same levels of the cord in different individuals, according to the more or less complete pyramidal decussation at the lower end of the medulla. There are strong reasons for regarding the fibres of this tract as chiefly destined for the upper extremities.

Crossed or Lateral Pyramidal Tract.—Constituted by the larger proportion of fibres from the anterior pyramids* which undergo decussation at this high level (see fig. 1, *py*), the lateral tract passes down in the posterior section of the lateral columns, becoming like the *anterior direct tract*, progressively diminished in size to the lowest level of the cord. The fibres of this tract pass into the grey substance of the anterior cornu between the two horns, to become connected with its motor nerve cells through the intervention of their terminal arborizations. The direct and crossed pyramidal tracts represent a continuous connection with the cortex of the motor area of the brain, passing uninterruptedly in this course through the **crusta** of the **cerebral peduncle** and the **internal capsule.**

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* In rare exceptions even less than half the pyramidal fibres decussate at this level (Flechsig).

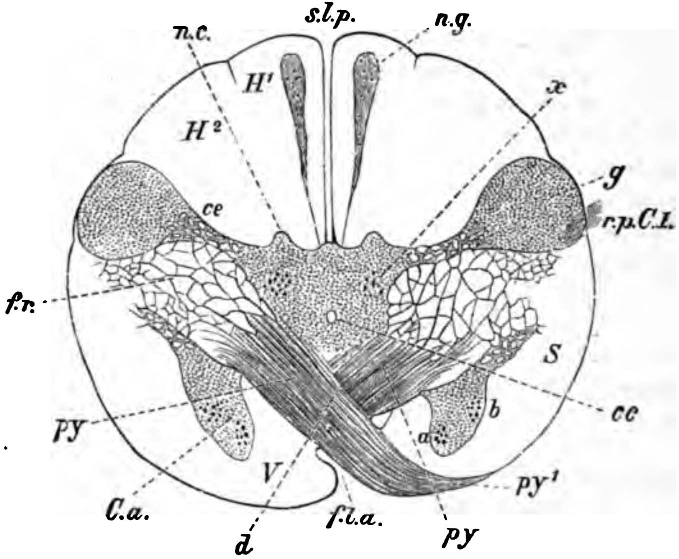

Fig. 1.—Section across transition region of medulla, showing decussation of pyramidal tract and nuclei of posterior columns.

s.l.p, Posterior longitudinal sulcus.
n.g, Nucleus funiculi gracilis.
n.c, Nucleus funiculi cuneati.
H^1, Funiculus gracilis.
H^2, Funiculus cuneati.
c.e, Neck of posterior horn.
g, Head of posterior horn.
r.p.C.L, Posterior roots of first cervical nerve.
x, Cell group in base of posterior cornu.
cc, Central canal.
S, Lateral column.
a and *b*, Cell clusters in anterior cornu.
C.a, Anterior cornu.
py, Pyramidal tract from lateral columns decussating at *d* as they ascend.
f.l.a, Anterior longitudinal fissure.
f.r, Formatio reticularis.

Posterior Median Column.—This wedge-shaped column of fine nerve fibres lying on each side of the posterior median fissure, scarcely recognisable below the dorsal region, extends from this site upwards to its termination in the **clavate nucleus** in the medulla. It increases steadily in size from below upwards, and undoubtedly receives fibres in part from the sensory nerve-roots which pass into this column by way of the postero-external column as well as the posterior commissure.

Postero-Lateral Columns cannot, like the postero-median, be regarded as largely a *continuous* tract throughout the spinal cord. A great part of their bulk is constituted by the inner division of the posterior nerve-roots which, curving round the caput cornu, run obliquely upwards or downwards, or directly outwards, to enter the grey matter of the horn; and also, in part, by a system of short, vertical, commissional fibres passing betwixt different levels of the grey matter throughout the whole extent of the cord. Higher up in the medulla we shall find that this column terminates in the **cuneate nucleus.**

Direct Cerebellar Tract.—A somewhat flattened fasciculus forming the marginal zone of the lateral columns, from the end of the dorsal region upwards, lies upon the outer side of the **lateral** or **crossed pyramidal tract;** its fibres arise from the **posterior vesicular column of Clarke,** which, as we have seen above, commences at the level of the third lumbar nerve. It gradually augments in size, and eventually terminates in the cerebellum, passing up to it along the restiform tract of the inferior peduncle. At its origin, and high in the cervical region also, the **lateral pyramidal tract** becomes superficial behind, so as to separate it from the posterior cornu.

Antero-lateral Ascending Tract (Gowers).—A column of fibres extending up through the whole length of the cord (occasionally the seat of ascending degenerative changes) has been described by Dr. Gowers as situated in front of the crossed pyramidal and direct cerebellar tracts. The tract is regarded as a sensory tract originating from root fibres of the sensory nerves *decussating* across the posterior commissure.* Arising in the lumbar region where it lies across the lateral columns on a level with the posterior commissure, it becomes placed, higher up in the cord, more superficial. Here, in transverse section, it forms a comma-shaped tract, its head lying betwixt the crossed pyramidal and direct cerebellar tracts, whilst its attenuated tail extends along the outer border of the cord, almost as far as the anterior median fissure. It can be traced up into the medulla in front of the direct cerebellar tract, but where the latter unites with the restiform body, its further course is not definitely ascertained. Some authorities (*Bechterew*, *Bruce*, and *Tooth*) suggest its termination in the lateral nuclei; others (*Hadden* and *Sherrington*) trace it into the restiform tract.

Anterior Radicular Zone and Lateral Limiting Layer.—These may be considered together as constituting, like many of the fibres of the posterior radicular zone, a series of short commissural fibres uniting the grey matter at different levels. In the case of the anterior root zone a certain portion of the fibres decussate at the **anterior commissure,** and thus "a connection may be established between the two anterior cornua at different levels." (*Gowers*[*]).

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* *Diagnosis of Diseases of the Spinal Cord*, First Ed., 1879; and *Diseases of the Nervous System*, vol. i., page 122 (Gowers).

We might summarise in the following short scheme the probable relationships of these tracts, as taught us by the Wallerian degenerations following upon disease or physiological experiment:—

Short commissural vertical tracts—
    (1) Anterior root zones.
    (2) Lateral limiting layer.
    (3) Burdach's columns (in part).
Descending motor from cortex cerebri—
    (4) Türck's columns.
    (5) Crossed pyramidal tract.
Ascending sensory tracts—
    (6) Goll's columns from posterior roots.
    (7) Direct cerebellar from visceral tract.
    (8) Antero-lateral ascending from crossed sensory roots.

THE MEDULLA OBLONGATA.

A transverse section taken just below the **calamus scriptorius** so as to reveal the central canal intact, ere it opens out on the free surface of the fourth ventricle, shows us the central grey matter thrust back to the posterior margin of the section—yet encroached upon laterally by the mass of the **clavate** and **cuneate nuclei** on either side. The central grey substance situated in the middle line is symmetrically disposed around the central canal, which here forms a mere elongated slit. Most prominent in front, it presents an eminence on each side of the median raphé, with a rich nucleus of large nerve cells, really disposed in double clusters—the nuclei of origin of the hypoglossal nerve, the fibres of which conspicuously run forwards towards the **olivary region.** On each side of these median prominences, a lateral projection of grey matter also occurs in front of and partially surrounding a conspicuous column of medullated fibres seen in transverse section encircled by medullated loops—the **solitary fasciculus, fasciculus rotundus** or **respiratory fascicle.** From this point the central grey matter inclines backwards to the middle line, behind the central canal, and at an acute angle to the former. This sudden inclination backwards is necessitated by the prominence of the **clavate nuclei,** which, lying behind the central grey substance and to its outer side, approach each other near the mesial line; along the backward inclination of this and the lateral prominence, nuclei for the origin of the accessory nerve and vago-accessory system are found. On each side of the central canal a column of fibres enclosing an elongated nucleus of nerve corpuscles, measuring $23\ \mu \times 11\ \mu$, extends forwards towards the middle line; and here, still enclosed in the central grey area, is a mesial compact cluster of small cells. The former represents the nucleus of the **eminentia** or **fasciculus teres,** which at higher levels becomes a prominent feature on the floor of the ventricle. Root fibres of the accessory nerve will at this level be traced from the lateral angle or eminence of the grey substance to their site of emergence behind the olivary body.

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[*] *Loc. cit.*, p. 123.

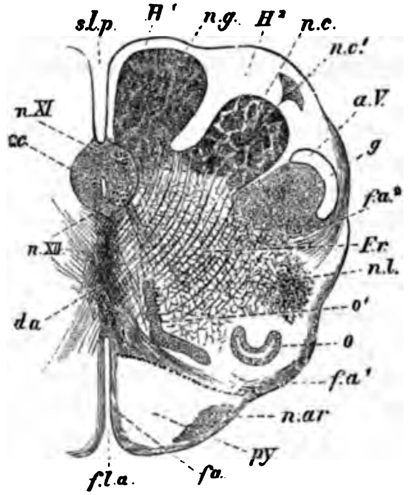

Fig. 2.—Cross-section of medulla oblongata at the upper decussation of the pyramidal tract.

s.l.p, Posterior longitudinal sulcus.
H^1, Funiculus gracilis.
n.g, Nucleus of funiculus gracilis.
H^2, Funiculus cuneatus.
n.c, Nucleus of funiculus cuneatus.
$n.c^1$, External nucleus of funiculus cuneatus.
a.V, Ascending root of trigeminus.
g, Substantia gelatinosa.
f.a, $f.a^1$, $f.a^2$, External arciform fibres.
F.r, Formatio reticularis.
n.l, Nucleus of lateral column.
o^1, Accessory olive.
o, Inferior olivary body.
n.ar, Nucleus arciformis.
py, Pyramid.
f.l.a, Anterior longitudinal fissure.
d.a, Anterior or upper decussation of pyramids.
n.XII, Nucleus and root fibres of hypoglossal nerve.
c.c, Central canal.
n.XI, Nucleus of spinal accessory.

Above the *calamus scriptorius*, the opening up of the central canal on the floor of the fourth ventricle, is necessarily attended by a recession of the clavate nuclei; and the **eminentiæ teretes,** which, as we stated, formed the antero-lateral boundaries of the central canal, become now exposed on the surface, as the innermost column seen in this lower half of the ventricle, on either side of the median raphé. Formerly covered by the **ala cinerea,** which represents the nucleus of the vagus, the *fasciculi teretes* pass upwards as white columns, strongly contrasting with the grey of the vagus nucleus; and, as they take this course, they lie superjacent to the hypoglossal nuclei. These eminences, therefore, map out the course of the hypoglossal nuclei, but must not be identified with that nerve, as they belong to a wholly distinct system. Whilst the white columns of the eminentiæ teretes become wider and more pronounced upwards, the ala cinerea disappears between them and a more external eminence—the **acoustic tubercle**—so that transverse sections exhibit on either side of the median raphé from within outwards the **eminentiæ teretes,** the **tuberculum acousticum,** and lastly the **restiform columns.**

At this plane the central grey matter is consequently unfolded outwards—a strongly marked concavity directed backwards, still characterises this region of the ventricle; but this process of unfolding proceeds at higher levels, until on a plane with the **striæ acousticæ,** the floor of the ventricle is almost flattened out, presenting only a gentle depression at the middle line. As this process of unfolding of the central grey matter proceeds, the restiform tract diverges more and more from the mesial line; whilst simultaneously the central grey substance, notably in the region of the hypoglossal nucleus, becomes shallower, and the nucleus itself nearer the surface.

Superficially viewed in the *fresh* medulla one readily sees between the diverging restiform columns a large central **V,** divided midway by the vertical raphé and separated from the plump acoustic tubercles on either side by a well-marked depression, into which the upper wedge-shaped apex of the *ala cinerea* plunges and loses itself. The anterior border of the grey substance now loses its abrupt prominences, and assumes a gentle sinuous course across the medulla from one solitary fasciculus to the other—the several wave-like summits representing the site of the **hypoglossal** and the **vago-accessory nuclei** (fig. 3).

Having so far followed the disposition of the central grey matter, from just below the opening-up of the central canal to the level of the striæ medullares of the acoustic nerve, the student should now direct his attention to a cross-section of medullated fibres of a notable crescentic configuration, and encircling on its outer side a coarsely reticulated region largely made up of deep stained connective tissue. These conspicuous structures lie laterally disposed near the margin, on either side, in all sections of the medulla up to the emergence of the trifacial nerve: the dark-stained reticulum is the representative of the **substantia gelatinosa** of the posterior cornu. The medullated crescent is the ascending root of the fifth nerve, representing the ascent of the remaining portion of the posterior root zone of the spinal cord. Drawing an imaginary line from the solitary fasciculus outwards to this crescent—to its anterior border in the lower levels, and to its posterior border in the higher level near the acoustic—we map off a region which corresponds to the posterior columns of the spinal cord, and their continuation as the inferior peduncles of the cerebellum: this region lies *behind* the imaginary line so drawn.

In like manner, a line drawn from the mesial eminence of the central grey matter obliquely outwards to the root of the inferior olivary body, maps off the remaining portion of medulla into two divisions, an inner, between it and the median raphé; and an outer, between it and the former line drawn to the crescentic root of the fifth nerve. The inner of the two divisions corresponds to the **anterior column** of the cord; the outer division, to the **lateral columns.** The two imaginary lines, so drawn, correspond to the direction taken by a

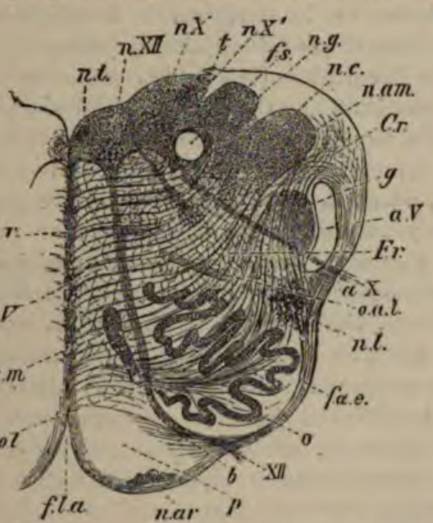

Fig. 3.—Section of medulla oblongata through the inferior olivary bodies.

n.t, Nucleus of fasciculus teres.
n.XII, Nucleus of hypoglossal nerve.
n.X and *n.X*^{1}, Nuclei of vagus.
f.s, Funiculus solitarius.
n.g, Nucleus of funiculus gracilis.
n.c, Nucleus of funiculus cuneatus.
n.am, Nucleus ambiguus.
C.r, Restiform tract.
g, Substantia gelatinosa.
a. V, Ascending root of trigeminus (fifth nerve).
F.r, Reticular formation of lateral column.
a.X, Root fibres of vagus.
o.a.l, Outer accessory olive.
n.l, Nucleus of lateral columns.
f.a.e, External arciform fibres.
o, Inferior olivary body.
XII, Emergent root fibres of hypoglossal.
p, Anterior pyramid.
n.ar, Nucleus arciformis.
f.l.a, Anterior longitudinal fissure.
p.ol, Olivary peduncle.
o.a.m, Inner accessory olive.
V, Anterior column.
r, Median raphé.

this lateral column of the medulla: one, which is the richer in cells, is elongated and directed from without inwards, approaching the margin of the lateral column, lying parallel with the roots of the lateral mixed nerves, between the *substantia gelatinosa* behind, and the **olivary** and its **fillet** in front.

Further inwards in this lateral column, and carried backwards parallel with the lateral mixed roots, is a second smaller group of cells more closely clustered than the former; not traversed, as these are, by dense fasciculi of arcuate fibres passing to the olivary body. The former called the **nucleus** of the **lateral column** by Stilling and Clarke, might be more conveniently termed the *external*, and the second cluster the *internal, nucleus* of the lateral column; or following Dr. Ross, the *anterior* and *posterior nucleus.*

They almost certainly represent motor cell-groups of the anterior cornua of the spinal cord, severed from the rest of the central grey matter by the decussation of the pyramids across to the lateral columns, and the interposition of the mass of the inferior olivary body. By Dr. Ross they are regarded as detached from the *antero-lateral* and *postero-lateral* group of cells in the anterior cornu by the cleavage effected by the arcuate fasciculi of the medulla, whilst those main groups are still found as the motor nuclei in the central grey matter of the medulla.

Intercalated between the lateral columns of the medulla and the anterior or median, is the *inferior olivary body*, extending throughout the region we have been studying, but terminating at the level of the lowermost fibres of the *pons*. Prominent on the superficial aspect of the medulla, between the pyramids and the lateral and restiform tracts, it looks like a small almond-shaped body, which upon transverse section reveals a grey nucleus, *imbedded* in a medullated investment of *longitudinal* fibres—the so-called **fillet** or **olivary fasciculus.** The grey nucleus is in the form of a plicated capsule of many folds, constituted of numerous cells imbedded in grey matter, and open towards its inner side. The investing medulla of longitudinal fibres passes inwards and forms a central core for this grey capsule, the fibres of which then spread out into its various convolutionary plications to terminate in the cells here distributed: the remaining fibres, which do not so turn inwards to the grey capsule, pass downwards into the lower regions of the medulla and cord. We have already alluded to the dense intertwining and connections of the cerebellar arcuate fasciculi within these olivary bodies.

In the lower planes below the calamus, our sections exhibit the olivary capsule open in front at its **hilus,** the anterior line of plications being shorter than the posterior: and here, bordering upon this opening in the capsule, is an elongated belt of grey matter, containing cells similar to those in the olivary body—this is the **internal accessory olive.** Sections taken midway through the inferior olive show us two such bodies; the one, as before, situated in the anterior column, separated from the olivary body by the root fibres of the hypoglossal, and greatly segmented by the passage of a rich system of arcuate fibres to the raphé: the other, in the lateral column just behind the hilus, like a concave lens with its concavity towards the olivary capsule—this latter is the **external accessory olive.*** In histological structure both resemble the larger olivary body, and by Meynert they are regarded as continuous with its capsule.

The *most external* arcuate fasciculi entering the restiform tract come to it by way of the anterior pyramid and olivary body, partly in front of and partly behind (and so encircling) the former, and forming a thick stratum of fibres over the external aspect of the olivary body—its **stratum zonale :** and lastly, covering in like manner, the ascending root of the fifth nerve to end in the restiform tract. A similar investment of the upper olivary body exists, as we shall see later on; this in the lower mammals is uncovered by the fibres of the pons, which do not conceal them, as in man, and constitute the so-called **corpus trapezoides.** The zonular layer passing over the anterior aspect of the anterior pyramids is aptly referred to by Meynert as a *small anterior pons.*

This inferior half of the medulla, with which we have for the present concerned ourselves, contains the nucleus of origin and emergent root fibres of but one purely *motor* nerve—the *hypoglossal*—but of four of the **mixed lateral system,** viz., the *spinal-accessory, vagus, glossopharyngeal,* and (the ascending root of) the *trifacial* or fifth. At the anterior mesial prominence of grey matter in these planes, we find the nuclei of the *hypoglossal,* which, prior to the opening up of the central canal, are arranged in a double cluster usually termed the *internal* and *external convolute* of the hypoglossal, owing to the loop-like arrangement of the centric and peripheric fibres connected with them. The external also lies at a posterior plane to the internal. The cells are large and multicaudate, forming the most conspicuous cell-groupings in the whole sectional area of the medulla: they measure .60 μ × 20 μ. The *centric* connections of these nuclei consist of certain straight fibres of the median raphé, which run backwards as far as the central grey matter, and then arching outwards, form spirals around the front and outer border of each nucleus, and are connected with its large cells: thence, similarly curving around the inner border to pass obliquely outwards, are the *peripheric fasciculi*—the *root-fibres of the hypoglossal.* These emerge from between the

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[*] These bodies are also known as the "external and internal parolivary bodies."

pyramid and the olivary body, some fasciculi traversing the latter in their course. In vertical extent this centre of origin stretches from just above the level of the decussation of the pyramids to the *striæ medullares* of the acoustic nerve; but, as we distance the *calamus*, the groups become less definite and merged into a less characteristic form, far less rich in cells. Throughout the whole of this extent, the vertical column of cells gives origin to emergent radicles, which issue anteriorly.

External to the hypoglossal nuclei lie the lateral projections of the central grey matter, in the angle of which we find the *sensory* nuclei of origin of the mixed lateral system of nerves, so named from their possessing both motor and sensory filaments. Some seven roots of origin are enumerated by Meynert for this system of nerves; and it is probable that the three nerves of this system in the lower half of the medulla arise in a very similar, if not identical, manner from closely associated nuclei, some of which are common to two nerves. The two more important nuclei of origin for this system are—the motor nucleus of the mixed nerves, and the sensory already alluded to.

The *motor nuclei* are found in advance of the central grey substance, disposed in the lateral columns of the medulla. A somewhat elongated cluster of large nerve cells, from which motor fasciculi emerge and run *backwards* parallel to the emergent root fibres of this system of nerves, is the more important of this mode of origin; but, fibres running in the same direction can also be traced further outwards, to the nucleus of the lateral column, between the ascending root of the fifth and the inferior olivary body. Much discrepancy appears with respect to the descriptions given to these anterior roots of origin of the mixed lateral system; some authorities speak of an anterior and posterior nucleus of the lateral column; others describe these fibres as being doubtfully *roots* of the system; whilst others with Meynert refer to one nuclear column of origin *distinct from the nuclei of the lateral* column. In fact, Meynert traces this motor nucleus as a nucleus of the spinal accessory downwards to the lateral process of the anterior horn, and finds its analogue on higher levels in the **inferior facial** and **motor nucleus** of the **trigeminal.**[*] Our own view of the case would be in accordance with that of Meynert; in addition to which, however, we would assign to the *external nucleus of the lateral column* a partial site of origin for these motor rootlets. The important fact for the student to bear in mind is that these motor nuclei are, in accordance with the spinal cornual scheme, in advance of the sensory division, and reach the main roots by recurrent fasciculi curving round the vagus nucleus from the inner to the outer side; and this type is repeated for the motor roots of the facial and trigeminal nerves.

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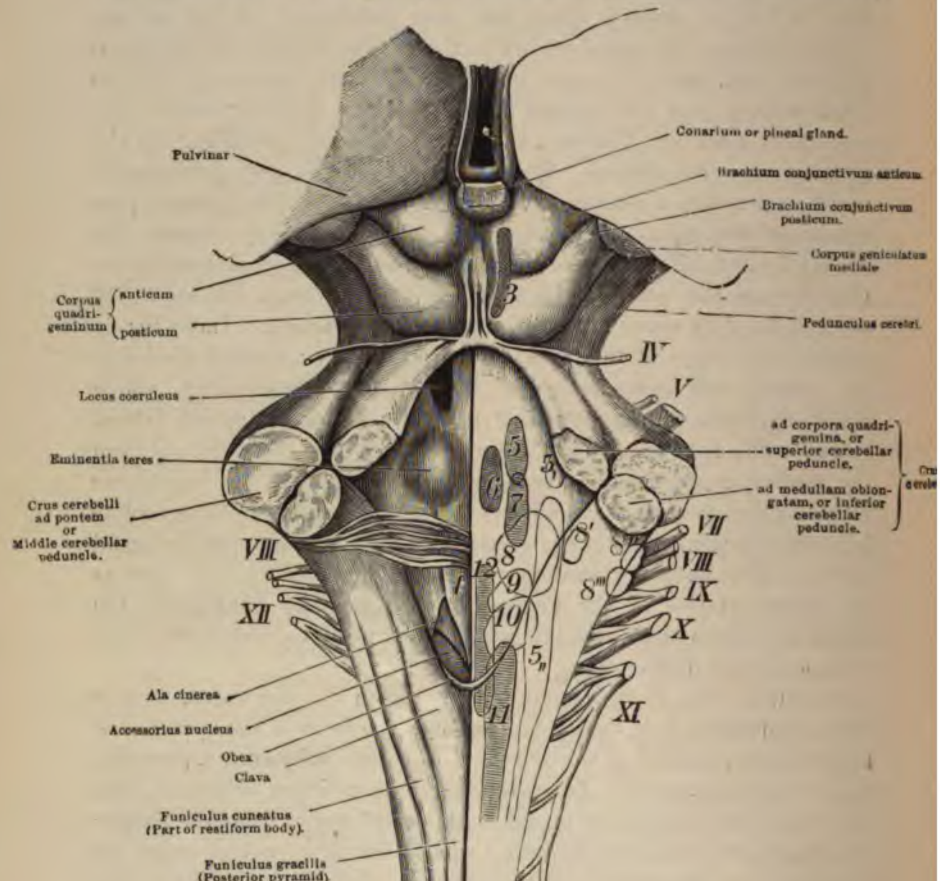
[*] *Psychiatry*, translated by Sachs, part i., p. 124.

The **sensory nucleus** or posterior nucleus for these three mixed nerves is a somewhat compact formation of nerve cells, clustered within the lateral angle of the central grey substance at the lower planes of this region, where it forms the vago-accessory nucleus. A little difficulty may be experienced in distinguishing between the nucleus of origin for the three nerves, if we do not attend to the fact that so long as arcuate fibres are seen distributed to the solitary funiculus from the raphé, we are in the region of origin of the spinal accessory and *below the vagal nucleus;* the latter centre can also be differentiated into two groups, an *external* and *internal nucleus*, described by Lockhart Clarke, a similar arrangement prevailing for the glosso-pharyngeal centres. From the apex of this grey prominence, which represents a sensory column of origin for these nerves, pass outwards the main root fibres—the spinal accessory *between* the olivary bodies and gelatinous substance; the vagus and glosso-pharyngeal *through* the latter, and traversing in their course the ascending roots of the fifth nerve ere they emerge at the surface. Immediately outside the origin of this root, at the posterior or sensory nuclei, is the conspicuous cross-section of the **solitary fasciculus**, which really represents an **ascending root** for the same mixed lateral system. We have seen that a dense arcuate system passes into it from the median raphé (*centric* fibres) below the origin of the vagus; we may also just as readily trace fibres issuing from this ascending root to join the emergent roots of the accessory, vagus, and glosso-pharyngeal nerves. The posterior sensory nucleus is regarded by Dr. Ross as the representative of the **vesicular columns of Clarke** in the spinal cord from their relative position, connections, character of the cells, and their distribution.

A fourth root easily traced in the region of the vagus, is one which, emerging from the raphé, traverses the front of the hypoglossal nucleus, and, following the curve of the grey substance anteriorly, enters the vagus nerve. In traversing the gelatinous substance also, the vagus and glosso-pharyngeus both derive fibres from the former ere they issue from the medulla; this is the fifth root of origin for these nerves. Another small fasciculus has been described by Clarke as passing from the **fasciculus teres** into the **vagus.**

The Upper Half.—Passing now to the *upper half* of the medulla, which upon its ventricular aspect is, like the lower half, triangular in outline, its base being mapped out by the acoustic striæ and its lateral boundaries formed by the superior cerebellar peduncles, converging to the quadrigeminal bodies, we meet first with *two motor nerves* closely associated in their origin, and arising, as do the motor cranial nerves generally, from an anterior or median position on either side of the raphé; and *one purely sensory nerve*, which takes its origin, in accordance with the same morphological principle alluded to, from a lateral and posterior plane. The two motor nerves are the *sixth* and *seventh* pair, or the **abducens** and **facial**; the sensory nerve is the *eighth*, the **acoustic** or **auditory.** The nuclei of origin for these three nerves do not occupy the same vertical plane; that for the sixth is the highest, next below it comes the facial nucleus, and lowest of all the acoustic nuclei—yet they each successively overlap the other, the internal acoustic nucleus, as we have already seen, descending also below the base of the arbitrary triangular space drawn by the striæ medullares (see fig. 4, *6*, *7*, *8*). As in the *lower* triangular area of the grey floor below the striæ, we found the nuclei of a motor (XII) and three mixed nerves (IX, X, XI) associated


Pulvinar
Conarium or pineal gland.
Brachium conjunctivum anticum
Brachium conjunctivum posticum.
Corpus geniculatum mediale
Corpus quadrigeminum anticum
Corpus quadrigeminum posticum
Pedunculus cerebri.
IV
V
Locus coeruleus
ad corpora quadrigemina, or superior cerebellar peduncle.
ad medullam oblongatam, or inferior cerebellar peduncle.
Eminentia teres
Crus cerebelli ad pontem or Middle cerebellar peduncle.
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VII
VIII
IX
X
XI
XII
VIII
Ala cinerea
Accessorius nucleus
Obex
Clava
Funiculus cuneatus (Part of restiform body).
Funiculus gracilis (Posterior pyramid).

Fig. 4.—Medulla oblongata and pons with neighbouring structures seen from behind: schematic representation of the nuclei of origin of the several cranial nerves.

through a great part of their extent with the accessory body—the **inferior olivary;** so in this *upper* triangular division we find a very similarly constituted structure—the **superior olivary body**—occupying a vertical plane corresponding very nearly to that of the two motor nerves—the sixth and seventh.

Transverse sections across the levels of emergence of these latter nerves exhibit a notable change in the distribution of the various structures in front; the inferior olivary bodies have disappeared; the pyramids still maintaining their integrity as *independent, compact columns*, are now concealed beneath the most anterior fibres of the middle cerebellar peduncle (*pons*), which enclose them between their transversely disposed fasciculi, as a more superficial and a deep series of fibres; whilst laterally the **brachia** of the pons diverge to the cerebellum, and further back the restiform and internal divisions of the inferior cerebellar peduncles in like manner pass to their distribution. On a level with the striæ medullares an intermediate transitional stage is apparent; and, as we pass to higher planes, the inferior olivary body loses its outward inclination, its long axis becoming disposed antero-posteriorly and immediately behind the two **pyramids.** Thus a lateral constriction occurs which gives the medulla here from before backwards an apparent but not absolute increased depth. This antero-posterior depth appears still more exaggerated by the lower loops of the pons capping the pyramids in front, which have just been caught at this plane and divided. In such sections the nucleus of the lateral column is still well seen between the diminished olivary body and the ascending root of the *fifth* nerve, whilst immediately posterior to the inferior olives is a group of large fusiform and multicaudate cells, the former in connection with the arcuate system here, the latter in apparent connection with fasciculi which pass backwards to the **median** or **motor column** of **grey matter.**

Still somewhat higher, the inferior olive ceases, or may present its upper extremity as a single minute plication; and, in this region, we find the nucleus of the lateral column compressed into a long narrow tract by the interposition between it and the ascending root of the *fifth* nerve of a very notable large nucleus of almost spherical outline, and, by the disposition of its enclosing fibres, severed apparently into a series of convolutes of large multicaudate cells. This is the **anterior** or **inferior nucleus** of the **facial nerve,** and from it a somewhat wide belt of sparsely scattered fasciculi pass back to ascend, as we shall see later on, as the **genu** of the facial nerve, whilst the compressed nucleus of the lateral columns sends indistinctly marked fibres towards the median grey. The superior olivary does not as yet present itself; in this plane we may study the various nuclei of origin of the auditory nerve. Following the grey matter of the floor of the ventricle outwards from the median prominence (which here is remarkably shallow), we find it progressively increases in depth to its extreme lateral limits, where the lateral or sensory projection is a notable feature, and the large **internal auditory** nucleus is seen. Immediately outside this sensory nucleus is the tessellated area characterising the *inner division* of the inferior cerebellar peduncle; followed still further outwards by the transverse section of the crescent-like *restiform tract*. To the inner side of the restiform tract the conspicuous ascending root of the *fifth nerve* is applied.

The whole of the structures above noted—the grey floor with its lateral prominence, the inner peduncular tract, and the restiform column—are embraced superficially by a zonular investment of fibres issuing from the region of the raphé; in fact, the *striæ medullares*, which, reinforced further on by others emerging from the restiform tract, constitute the posterior root of the auditory nerve.

On the other hand, these same structures above enumerated, are embraced *from within* by the anterior auditory root, which runs chiefly between the restiform tract and the ascending root of the fifth, although many of its fasciculi traverse the structure of the latter. The student should remark here that the fifth ascending root serves always to distinguish to him the emergent roots of the *facial* from those of the *auditory*; the facial lying to the inner, and the auditory to the outer side of this root.

We should, therefore, regard the auditory nerve as possessing two roots of origin—(1) anterior, also called the internal, deep or vestibular root, whose peripheral destination is the semi-circular canals; and (2) the posterior, also called the external, superficial or cochlear root, which in its turn ends in the cochlea.

Posterior Root.—The fibres from this source enter the anterior or accessory auditory nucleus, which forms so prominent a feature as it lies upon the deep root of the auditory nerve, and, as suggested by Bruce, might much more justly be termed the **auditory ganglion.**

From this centre they form connections with the following structures:—

(*a*) Through the striæ acousticæ with the **formatio reticularis**.

(*b*) With external and internal acoustic nuclei of *same* side.

(*c*) With the flocculus.

(*d*) With superior olive of the same and opposite sides by the medium of the trapezoid body.

Anterior Root.—The vestibular root, continuous with the so-called ascending root of the auditory nerve, arises in part from the cuneate nucleus, in part from the large cells of the external acoustic or Deiter's nucleus; whilst a further division passes into the internal or **chief** nucleus of the auditory; and some fibres are traced into Bechterew's nucleus. From the internal and external acoustic nuclei the following connections are traced:—

(*a*) With the sixth nerve nucleus of the same side.
(*b*) With the inferior olivary of the same side.
(*c*) With the flocculus of the same side.
(*d*) With the opposite formatio reticularis.
(*e*) With the opposite roof nuclei of the cerebellum.
(*f*) With the opposite posterior longitudinal fasciculus and thus possibly with the third nerve nucleus.

Lastly, the cuneate nucleus may bring the vestibular root into connection with the fillet and with both restiform bodies (*Bruce*).

At higher planes of the medulla wherein the superior olivary body appears, we reach the radicular zones of the **facial** and the **abducens.** The *motor area of the grey floor* of the ventricle at these levels presents in transverse sections, two strongly defined eminences separated by the median groove and raphé—these are the eminences over the facial genu and the nucleus common to both facial and abducens nerve. The *sensory area of the grey floor* flanks these eminences on either side like walls, diverging from them at a somewhat obtuse angle, the enclosed space being bridged over by the cerebellum.

On either side of the median line at the extreme posterior end of the raphé, is an oval cross-section of medulla 1¼ mm. by a ½ mm. in size, sharply defined and lying between the grey matter of the floor and the *hindmost series* of arcuate fasciculi given off from the raphé; it represents the root of the facial nerve in cross-section at its curvature upwards, otherwise called the **facial genu.** From its neighbourhood medullated fasciculi sweep in a wide curve, following the inner margin of the grey matter as far as the sensory area, when they pass forwards and outwards to their emergence from the medulla, forming in this latter course the boundary between sensory and motor divisions. In the sensory division outside this root lies, as we before indicated, the **ascending trigeminal root.** The sweep of the facial in its course beneath the grey floor encloses a large and important nucleus, measuring 3 mm. in widest diameter, very rich in cells which are multicaudate, and are disposed in an almost circular area; from the outer side of this nucleus emerge root fasciculi, which are distinctly seen to join the facial as it sweeps forwards round the nucleus to its point of emergence.

On the other hand, from the posterior and inner margin of this nucleus other fibres emerge, which upon the inner side strike forwards, becoming gradually more divergent from the raphé in sharply defined fasciculi, to leave at the lowest border of the pons as the **abducens**

nerve or sixth pair. The nucleus itself is the **abducens-facialis,** also termed the **posterior** or **superior facial nucleus,** or again the **nucleus of the sixth nerve.** It is placed in communication with the cerebrum by means of the fibræ rectæ of the raphé, which can be readily traced as the most posterior of the arcuate fibres curving first around the facial genu in front, and then passing round the lower hemisphere of the nucleus. Considerably in front of the abducens-facialis nucleus, and in the motor division of this region, lies the **inferior** or **anterior facial nucleus,** almost parallel with the trigeminal root, but separated from it by the facial emergent fasciculi; its fibres pass backwards, as we have already seen, at lower levels to

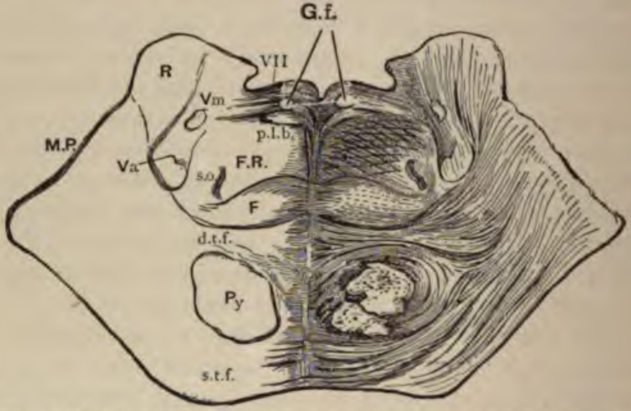

Fig. 5.—Section through pons on a level with the origin of the great root of the trigeminus.

G.f, Genu of facial nerve.	*F.R*, Reticular formation.
VII, Root fibres of facial.	*s.o*, Superior olivary body.
Vm, Motor nucleus of trigeminus.	*F*, Lemniscus or fillet.
Va, Ascending root of trigeminus.	*Py*, Pyramids.
R, Restiform tract.	*s.t.f*, *d.t.f*, Superficial and deep transverse fibres of the pons.
M.P, Fibres of pons varolii.	
p.l.b, Posterior longitudinal fasciculus.	

arch beneath the abducens-facialis to its inner and posterior aspect, and thence running upwards as the *genu* of the facial, again bend around its upper border in the graceful sweep of the emergent roots.

The Superior Olivary Body.—We have seen that this body extends from the lowest border of the *pons* through the whole tract of origin of the facial nerve, being well-exposed in cross-sections, lying between the inferior facial nucleus and the emergent root fibres of the sixth nerve. The transversely disposed fasciculi lying upon its anterior surface, extending from the decussation at the raphé to ascend in the inferior cerebellar peduncle, form the so-called **corpus trapezoides** which becomes exposed superficially in animals where the dimensions of the pons are greatly reduced with the diminished supply of fibres reaching the medulla from the crusta. The **lemniscus** or **fillet** lies in these planes to the inner side of the superior olive, forming the pale-stained area of truncated triangular outline next the raphé, the base traversed by the most posterior fasciculi of the pons and **trapezoid** formation. Into the fillet at higher planes, fibres of the upper olive pass to be connected with the central grey of the lower quadrigeminal body, the **testes**; functionally these fibres should be regarded as centrifugal, since they have been found by Flechsig to *degenerate downwards* to the superior olive.

A *cerebellar* connection is established between these bodies and the roof nuclei (nuclei tecti) of the middle lobe of the cerebellum; whilst other fibres pass back from them also to the nucleus of the sixth and of the auditory nerve, as well as to the lateral columns of the spinal cord. Motor impulses, therefore, emanate from this body to the sixth nerve nucleus, which, being connected by decussating fibres with the nucleus of the opposite **motor oculi,** subserve the conjugate deviating movements of the eyeballs. In like manner, motor impulses to the lateral columns of the cord explain the associated movements of the head to the same side.

The quadrigeminal bodies, on the other hand, which are connected with the optic tracts, transmit stimuli thence emanating, to the superior olivary bodies through the medium of the fillet, and so to the oculo-motor apparatus of the sixth and third nerves.

We have already seen that the posterior columns of the cord resolve themselves through the intermediation of their clavate and cuneate nuclei into the restiform tract of the cerebellar peduncle. They also by the **anterior sensory decussation** of a portion of their arcuate fasciculi pass upwards on either side of the median raphé as the fillet, and thence to the quadrigeminal bodies. This portion of the fillet, it will be observed, is a *centripetal* or *sensory tract*, so that the fillet really contains systems of ascending and descending fibres, as is indicated also by the results of lesions affecting the tract.

At the level of origin of the sixth and seventh cranial nerves, the central grey forming the floor of the ventricle is, as we have seen, extended laterally, shallow from before backwards, and bounded on either side by the restiform tracts: as we ascend to a higher plane we find the superior cerebellar peduncle on each side, which, in their descent, restrict the lateral extension of the ventricle and its investing grey substance. This occurs in such sections as are carried through the emergent roots of the fifth nerve. If we now follow the ventricle towards its upper angle, we find with the convergence of the superior peduncles towards the quadrigeminal bodies, the following changes in the disposition of the central grey:—First, the ventricle becomes narrower; the prominent lips of the grey matter become more pronounced, from the increase in the thickness of this formation; and at the same time the ventricle is roofed over posteriorly by the **anterior medullary velum.** The nuclei of the fifth pair alone of all the cranial nerves characterise this plane.

Higher still, we come upon the root fibres of the fourth nerve which decussate across the **aqueduct** posteriorly. The central grey here forms two notable protruding lips on either side of the mesial line, converting the aqueduct into a Y-shaped figure. Progressive thickening of the central grey substance occurs as we carry our sections through the posterior and anterior quadrigeminal bodies; the aqueduct restricted in size is completely surrounded thereby, and suffers minor alterations in its outline until it opens up into the central cavity of the third ventricle. From the level of the crossing of the fourth nerve upwards, the Y-shaped grey exhibits the *beak* of the Y interposed between the two notable bundles of the **posterior longitudinal fasciculus,** behind which we may continuously follow an *anterior* or *motor* column of grey matter, containing nerve cells, and externally a *lateral* or *sensory* column, such as characterised the cranial nerve origins in the lower half of the medulla. Betwixt these planes and the upper roots of the facial nerve lie the emergent fasciculi of the fifth nerve, which, in accordance with its mixed motor and sensorial function, also assumes a lateral site of origin. We will now take these upper cranial nerves *seriatim.*

The fifth or *trigeminal* has the nucleus of origin for its *motor root* within the motor area of the pons somewhat similar in position to the nuclei of the lateral division, which at lower levels sent fasciculi to the mixed system of nerves—the so-called **nucleus ambiguus.** It, however, lies considerably behind this nucleus of the facial—the anterior facial nucleus—and cannot be mistaken for it, since it does not present the same convoluted structure; and, moreover, is not in the mid planes of section of the upper olive, although the upper end of this structure is still seen. Its centric fibres pass to it from the median raphé where they decussate.

The origin of the sensory root of this nerve is far more extensive. We have throughout the whole of the medulla followed up in our sections the **ascending root** of the **trigeminal,** noting how in the lower planes of the medulla the vagus and glosso-pharyngeal, and higher up, the facial, traversed its cross-section near their points of emergence, and now find it lying between the motor nucleus and the restiform tract in the sensory area of the pons, throwing forwards its root fibres to emerge between the transverse fasciculi of the pons. Below, this *ascending root* appears to end in the tubercle of Rolando, and so would seem to have a close connection with the **caput cornu**

posterioris. A median root is described as originating from a nucleus at the level of emergence of the sensory root, in contiguity with the ascending root and the motor nucleus. On the lateral margin of the central grey around the aqueduct, as high as the anterior quadrigeminal body, or **nates,** we find the cross-section of the descending root of the fifth nerve with very characteristic spherical or vesicular cells lying in the central grey upon the inner side of the root fasciculi. Both cells and descending fasciculi become more conspicuous at lower levels, and the latter extend to the level of the exit of the sensory root, where they join it to emerge from the pons. Internal to this descending root is a series of deeply pigmented nerve cells, forming the substantia ferruginea, which is seen through the grey floor of the ventricle at the site named the **locus cœruleus.** From these cells, according to Meynert, pass root fibres to the opposite trigeminal root (sensory trunk), which in their course surround and traverse the **posterior longitudinal fasciculus,** decussate at the posterior extremity of the raphé, and thence, following out the anterior margin of the central grey, arch into the opposite sensory root. Associated with these latter fibres are described others which issue from the median raphé posteriorly, and after decussation terminate in the sensory root likewise. Lastly, a **cerebellar root** is described by some authorities.

In the motor column of the central grey, lying immediately behind the posterior longitudinal fasciculus, on either side of the median line and beneath the *nates*, is a well-defined grouping of cells, which, however, usually presents an apparent segmentation into distinct clusters. These nerve cells, commencing as high up as the posterior commissure, are at first somewhat scattered, but assume a more compact form as they run backwards towards the upper half of the testes, in which region they appear lodged in a hollow of the posterior wall of the longitudinal fasciculus. This nuclear column represents the origin common to the **oculo-motor** (third) and the **trochlearis** (fourth) nerves; the upper, scattered, segmented portion is the nucleus of the *third* more especially; the lower compact segment lying at the junction of the two quadrigeminal bodies is the nucleus of the *fourth* nerve. Both are believed to receive their centric fibres through the median raphé, those of the third nerve decussating ere they reach their nucleus.

The segmentation of the nucleus of the motor oculi nerve has been variously described by different authorities: we follow here more strictly the account given by Bruce,* which certainly accords most

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* "On the Segmentation of the Nucleus of the Third Cranial Nerve," Alex. Bruce, *Proc. Roy. Soc. Edin.*, 1889. *Illustrations of the Nerve Tracts of the Mid and Hind Brain*, Alex. Bruce, 1892.

closely with our own observations. The whole nucleus consists of seven chief segments—a central with three lateral groups on either side, and of six minor segments, which we shall speak of as the superior, the inferior, and the external nuclei (see fig. 6).

The central or median nucleus lies along the mesial longitudinal plane behind the raphé, and consequently in the V-shaped interval betwixt the two posterior longitudinal fasciculi. It is freely connected by commissural fibres with the nucleus on either side of it, C.

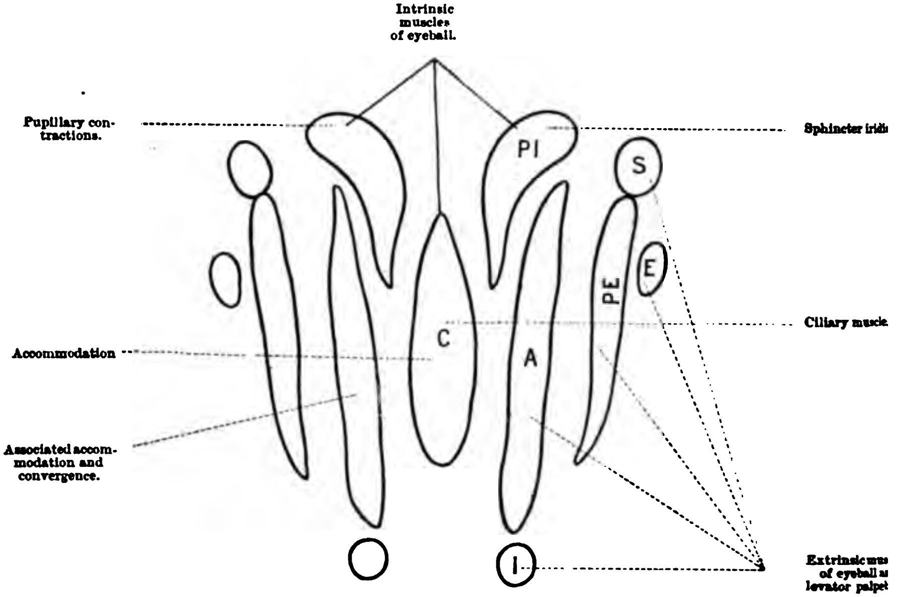

Fig. 6.—Scheme of segmentation of third nerve nuclei showing presumed anatomicla and physiological relationships.

A, Anterior nucleus; *C*, Central or median nucleus; *E*, External nucleus of Bruce; *I*, Inferior nucleus (segment of anterior); *S*, Superior or nucleus of Darkschewitsch; *PI*, Postero-internal or pale nucleus of Edinger-Westphal; *PE*, Postero-external or postero-lateral nucleus.

The anterior nuclei. On either side of the latter nucleus, and resting in the hollow of the posterior longitudinal fasciculus, is an important collection of nerve cells, the longest of all the oculo-motor segments, and most richly supplied with commissural fibres, coupling not only these two nuclei together but also connecting them with the intervening or central nucleus, A. These two anterior nuclei extend upwards and downwards beyond the levels of the central nucleus. The upper ends of these nuclei are but very scantily supplied with commissural fibres.

Postero-internal nuclei, also called the pale nuclei or nuclei of Edinger-Westphal, are wedged in between the central and anterior nuclei; narrow below and increasing in bulk upwards they terminate in a characteristic club-shaped head above the level of the anterior segments, PI.

Postero-external nuclei, also called the postero-lateral group, lie still more external along the outer divisions of the posterior longitudinal fasciculus, their nerve cells resembling those of the anterior group, PE.

The six minor segments consist of—(*a*) a superior couple; (*b*) an inferior couple; and (*c*) an external couple.

The superior, also called the nucleus of Darkschewitsch, is placed above the upper end of the postero-external nucleus, forming the extreme upper limit of the motor oculi nucleus, S. Bruce states that it forms the terminus for many of the fibres of the posterior commissure.

The inferior is just as clearly segmented off from the lower end of the anterior nuclei, and is distinguished from the latter by the complete absence of inter-commissural fibres, I.

The external is a group described by Bruce lying upon the outer aspect of the postero-external nuclei, and of much more limited extent than the latter group, E.

All these nuclei are intimately connected with the posterior-longitudinal fasciculus on the one hand, and with the root fibres of the third nerve on the other. So far as we can at present suggest the function of these several nuclei we may regard it as probable that the central subserve the function of accommodation; the postero-internal that of contraction of the iris; whilst the anterior is the centre for the associated act of accommodation with convergence (*Bruce*).

The root fibres of the *third nerve* emerge in a series of arched fasciculi directed forwards, with their concavities, for the most part, looking towards the raphé; and in their course traverse and partially encircle the **red nucleus** of the **tegmentum** which, as we shall see further on, lies on either side of the raphé above the plane of their decussation.

The root fibres of the *fourth nerve* take a much more circuitous course, running backwards instead of forwards to their point of emergence; an anomaly accounted for by Dr. Ross by the decussation of the upper cerebellar peduncles which occurs in the region of the testes severing the nuclear segment of the fourth from that of the third nerve, and so "compelling the former to seek its destination by an independent route."* Commencing at the junction of the nates and testes from this compact segment, the root fibres of the fourth nerve curve around the outer zone of the central grey matter to reach the anterior medullary velum, where they bound the aqueduct posteriorly. To reach this point, which lies below the testes, the root fibres must necessarily have traversed the full extent of the former, passing obliquely backwards and downwards to the **valve of Vieussens.** Crossing in the substance of this valve the fibres of opposite sides decussate, presenting another anomaly—since none of the other cranial nerves (except the optic tracts) decussate on the distal or peripheral side of their nuclei of origin, but invariably on their proximal or centric side.

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* *Diseases of the Nervous System*, vol. ii., p. 44.

THE MESENCEPHALON.

Upon the most casual examination of the cerebro-spinal axis, one is struck forcibly by the simple arrangement and solidarity of the spinal as compared with the remaining portion or upper end of this system—the gradual increasing complexity of the grey and medullated tracts of the **after-** and **hind-brain,** the so-called **medulla oblongata** and **pons,** and the uniform *divergence* of these tracts at higher and still higher levels: both gangliated masses and medullated systems alike severing their alliance, and diverging on either side in correspondence with the severance of the uppermost system into the two great hemispheric masses. As we rise step by step from medulla to pons, from pons to corpora quadrigemina, thence to the thalamic region, and lastly to the corpora striata of the **prosencephalon,** we find the grey masses becoming larger and more widely separated; whilst the medullated tracts, in like manner, diverge and empty themselves at different levels into the several ganglionic masses—in all cases, probably, to take a fresh departure to their final destination in the cerebral cortex.

Leaving for the time the more consolidated tracts of the **epencephalon** or **hind-brain,** and concentrating our attention on the **mid-brain**—*i.e.*, the **corpora quadrigemina** with the **crusta**—and the central grey common to the whole cerebro-spinal system, we find that the crura cerebri, athwart which the quadrigeminal bodies are placed, have two distinct tracts of wholly different destiny—the **tegmentum** and **crusta,** best seen in transverse section.

The former occupies the posterior and inner region of the crura, and partly empties itself into the superjacent quadrigeminal bodies, and in part into the **optic thalami:** whereas the latter—the crusta, lying in front and to the outer side of the tegmentum—becomes more and more divergent from its fellow of the opposite side as it courses upwards, passes in part into the basal ganglia, and in part expands into a wide-spread fan of fibres for distribution to the extensive cortex of the cerebral hemispheres. Thus we observe that the medullated tracts of the tegmentum and crusta in turn empty themselves into the several ganglionic masses met with from time to time—the **quadrigeminal bodies, pineal gland, thalami optici, striate**

bodies, and **cortex cerebri.** It is important that the student understand that the termination of the fibres upwards occurs in grey masses placed at different relative levels, answering to the position of the hind-brain, the mid-brain, the inter-brain, and the fore-brain: and he should gain a clear conception of each individual tract, so far as at present known, to its terminus in a grey centre. To return to the **mesencephalon,** the hind and inner portion of the crura forms, as we said, the tegmentum, and on this structure rest the quadrigeminal bodies and thalami; on the other hand, if we follow the crusta upwards, *as exposed at the base,* we find that each crusta diverges more and more until it meets the embracing **optic tracts,** which at this part of the base define the boundary between mesencephalon behind and **thalamencephalon** in front. At this point each crusta plunges deeply into the brain, disappearing between **thalamus** and **lenticular nucleus,** and passes up as a compressed medullated tract between the basal ganglia, emerging above their level as a wide-spread fan of fibres to the hemispheres. In this course, where they form a divisional wall between the large ganglia, they constitute what is called the **internal capsule,** the formation of which merits careful study.

If we imagine the crusta as seen from the base pass up unchanged in direction into the internal capsule, we shall then perceive that the latter would take a direction sloping obliquely upwards and outwards, presenting two surfaces—a lower, looking downwards, outwards, and backwards, roofing over the lenticular nucleus, and corresponding and continuous with the superficial surface of the crusta as far as the pons; and an upper surface, upon which the thalamus rests, corresponding to the deeper portion which lies adjacent to the tegmentum. It would also present two free borders—the internal or mesial, and the external or posterior. In the further expansion of this belt of fibres, the inner or mesial suffers displacement through the intrusion in mesial planes of the **head** of the **caudate nucleus,** whereby the anterior portion of the capsular fibres is thrust outwards, forming a sharp bend or "**knee**" with the posterior division. Thus we observe that the internal capsule is a stratum of fibres with a concavity looking downwards and outwards, arching as a roof over the lenticular nucleus, and forming a medullated bed, upon the anterior segment of which rests the caudate nucleus, and upon the posterior margin of which rests the thalamus.

If now we examine these ganglionic masses from above, as seen within the lateral ventricles, it is evident that the long axis of each is similarly disposed—*i.e.*, from the mesial line obliquely backwards and outwards; that the outermost of these masses, the two caudate nuclei, have their large pyriform head directed anteriorly, whilst their attenuated tail-like process appears pressed outwards by the narrow anterior pole of the wedge-shaped thalami. In like manner the latter,

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which have their broad extremity hindmost, are also pressed outwards behind by the intervening **quadrigeminal** and **geniculate bodies.** These three important structures—the corpora quadrigemina, thalami, and caudate nuclei—which represent the three divisions of the mesencephalon, diencephalon, and partly of the prosencephalon, as before indicated, rest upon and embrace the tegmental and crustal divisions of the cerebral peduncle.

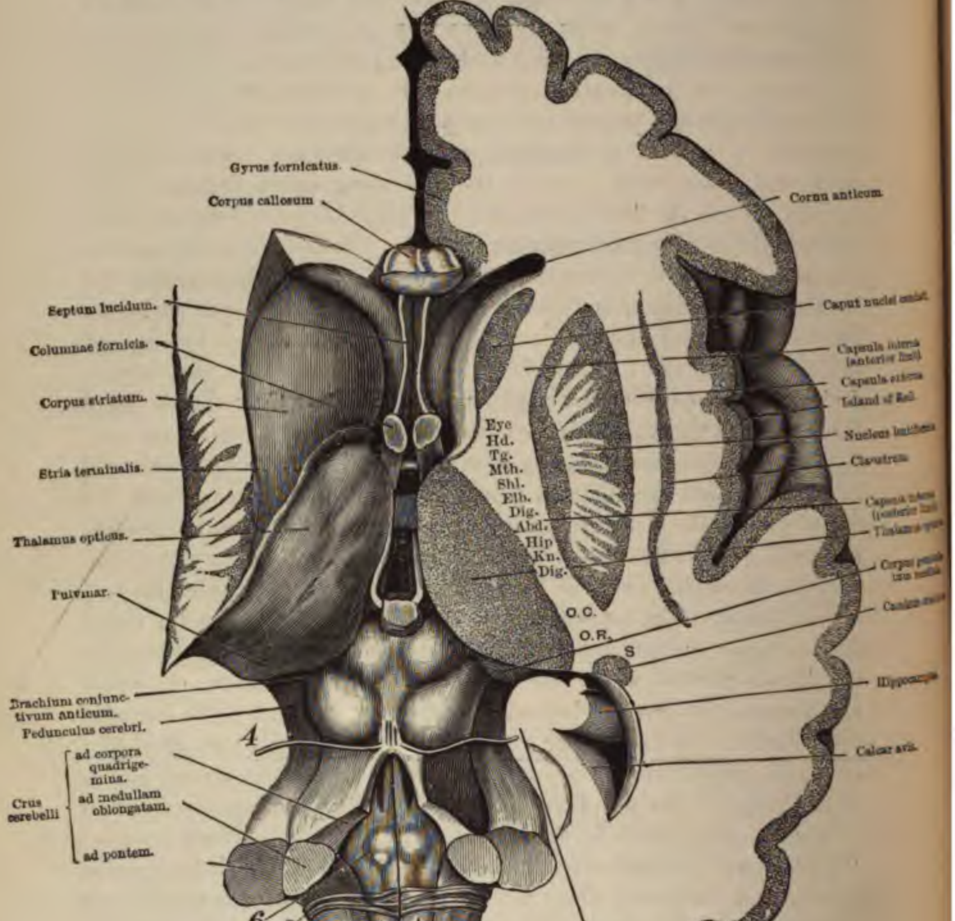

Fig. 7.—Horizontal section through hemispheres, the right at a deeper level than the left.

On the other hand, the outer division of the striate body—the lenticular nucleus—has its base directed forwards and outwards parallel with the **insula,** whilst its wedge-shaped apex is directed backwards and inwards towards the base—its upper surface, convex from before backwards, being adapted to the concavity of the internal capsule. The general relationships of the internal capsule are well seen in horizontal sections carried across the hemisphere at successively higher levels. Near the base it forms a quadrilateral section directed from within outwards and separating the tegmentum from the lenticular nucleus, having immediately behind it the **locus niger,** and parallel with it in front the **anterior commissure** as it passes outwards through the lenticular nucleus. It will be recalled that the crust, ere it disappeared into the depths of the hemisphere at the base, was bounded by the broad optic tracts: these are destined to terminate in the **external geniculate body,** and at a slightly higher plane these bodies appear immediately behind the extremity of the internal capsule as seen in transverse section. At a higher level, however, above the anterior, and through the plane of the **middle commissure,** the anterior portion is bent outwards, forming an obtuse angle or "knee," so that a horizontal section through *both* hemispheres at this level represents both internal capsules as an X-shaped figure with two anterior and two posterior segments (fig. 7). In the lateral angle of this X the wedge of the lenticular nucleus insinuates itself; between the anterior segments of the X the nuclei caudati appear; whilst the posterior segments include the optic thalami. At the central junction of these limbs, we find the fornix cut across.

Lastly, turning our attention again to the base of the brain, we see that the divergence of the peduncles, as they ascend, leaves between them in the middle line, first, the **posterior perforated space**—a bridge-like extension of grey substance at the angle between both peduncles, perforated by numerous vessels which enter the base of the thalamus at this point; and from which white fibres emerge and course round the upper margin of the pons—the **tænia pontis.** In front of the perforated space are two rounded bodies—the **corpora albicantia**—round which the **pillars** of the **fornix** turn; and from these bodies a thin grey lamella stretches forwards as far as the optic commissure, forming the floor of the third ventricle: from its floor a hollow tube—the **infundibulum**—descends, to which is attached the **pituitary body.**

Having thus far defined the limits of crusta and internal capsule, we can the more clearly appreciate the course and distribution of their fibres. Since the crusta represents the continuation upwards of the **pyramidal tract** of the cord, it must receive a considerable reinforcement of fibres within the pons to account for the much larger size which the crusta bears to the corresponding anterior pyramid of the medulla. An examination of the pyramidal tracts and crusta in a nine months' embryo gives us most valuable information respecting the origin of the fibres found therein. In the pyramids the outer portion of the tract consists of distinctly medullated fibres; the inner and anterior of non-medullated fibres; and an intervening portion contains a mixture of both. In the pons a similar distribution of these fibres is seen: whilst higher up still in the crusta, the non-medullated fibres occupy the inner third; the medullated fibres, the middle third; whilst the admixture of both is found behind and between the two former. This embryological dissection maps out three distinct systems of fibres which have received the respective names of (*a*) the **fundamental** or **medullated**; (*b*) the **mixed system**; (*c*) the **accessory** or **non-medullated system.**

Still further outwards, occupying the outer fourth of the crusta, just beyond the fundamental system, are found the fibres which represent the continuation upwards to the hemispheres of the sensory columns of the cord.

If we now trace these tracts upwards into the internal capsule, we find that the accessory or innermost passes up along the anterior segment of the capsule; the mixed occupies in its ascent the anterior third of the posterior segment, the fundamental the middle third of the same segment; whilst the posterior third or outermost part of the capsule is occupied by the sensory tract of the peduncles: or tabulated also with reference to their distribution, thus :—

TRACTS.	POSITION IN CRUSTA.	IN INTERNAL CAPSULE.	CORTICAL TERMINI.
Accessory Tract,	Inner third,	Anterior segment,	Third frontal gyrus posteriorly. Ascending frontal and parietal gyri at their lower end.
Mixed Tract,	Behind and between Accessory and Fundamental,	Posterior segment, Anterior third,	Second frontal and middle portion of central gyri.
Fundamental Tract,	Middle third,	Posterior segment, Middle third,	First frontal posteriorly. Upper end of central gyri; paracentral and parietal gyri.
Sensory Tract,	Outer fourth,	Do., do., Posterior third.	Temporo-occipital regions.

The above table reads as follows:—The accessory tract occupies the inner third of the crusta, passes up the anterior segment of internal capsule, and terminates in the cortex of the third frontal and lower end of ascending frontal and parietal gyri—and so for the remaining tracts.

The student must bear in mind that the crust of the peduncle contains a very large system of fibres, and that the tracts above described by no means represent the whole series. Thus each crusta includes, not alone tracts of the pyramidal fibres, but the sensory tracts of the cords—fibres to the thalamus and caudate nucleus, &c. The tracts we have now traced have *no connection* with the basal ganglia: they pass between the lenticular nucleus, thalamus, and caudate nucleus *uninterruptedly* to their cortical termini. Thus, as we have seen, the pyramidal tract extends uninterruptedly between the cortex of the **central gyri** and their immediate neighbourhood, and the **motor** cells of the **anterior cornua** of the cord at different levels throughout its course: and this pertains both to the motor or pyramidal and to the sensory tracts. It is obvious that the body and lower extremities have the least specialised movements, just as the arm and hand and the muscles of articulate speech are excessively specialised; and, that whereas the accessory group of fibres which supply the latter chiefly have to deal with very complex and specialised co-ordinations, the muscles being very numerous, but relatively very small in size: the fundamental group which supply the large muscles of the limbs, and especially of the lower extremity, have to deal with simple massive movements, the musculature being correspondingly bulky and less specialised. We see, therefore, that the accessory group, arising from the cortex at the lower end of the central gyri, pass chiefly to the spinal levels of the face, mouth, and hands, whilst the greater bulk of the fundamental group arising at the upper end of the central gyri extend as far as the lumbar enlargement for the musculature of the legs. Hence the former constitute comparatively *short* loops, and supply *many* though *small* muscles; the latter constitute *very lengthy* loops, and supply the *largest* muscles of the frame. The latter originate in the largest nerve cells discoverable, the former or accessory in cells of greatly reduced dimensions. The fibres of the *sensory columns of the cord*, according to Meynert, undergo a decussation upon a level with the decussation of the crossed pyramidal fibres by arching forwards around the central grey column, and after decussation passing up on the outer side of the anterior pyramid to the hinder third of the internal capsule, when they suddenly turn back, and are distributed to the **occipital** and **temporo-sphenoidal lobes.** As before stated, they have no connection with the thalamus or lenticular nucleus. So far for the *direct* sensory and motor fasciculi of the crusta and internal capsule. The internal capsule, however, includes several other systems of fibres, of which the following are the more readily followed out:—

(*a*) Fibres to the cortex from the outer surface of the optic thalamus.

(*b*) Fibres to the cortex from the outer surface of the caudate nucleus.

(*c*) Fibres to the cortex from the upper and inner surface of lenticular nucleus. (Denied by Wernicke.)

(*a*) Thalamic radial fibres given off from the whole length of the outer surface of the thalamus radiate forwards, outwards, and backwards; the anterior radiations towards the frontal lobe; the median, outwards to the parietal; and the posterior, arching backwards and upwards to the occipital lobe. The latter or posterior is a very important formation, and is separately distinguished as the **radiations of Gratiolet:** it serves as the means whereby the several roots of the optic nerve, which terminate in the quadrigeminal, geniculate bodies, and thalamus are connected with the **occipital cortex.** These optic radiations pass through the posterior third of the internal capsule, and are consequently brought in close relationship to the **sensory peduncular tract** already described as occupying this position.

(*b*) In like manner, fibres radiate from the outer surface of the caudate nucleus to reach the cortex in planes internal to and above those of the thalamus.

(*c*) Fibres arise from the upper and inner surface of the lenticular nucleus to interlace with the fibres of the internal capsule.

(*d*) A system of fibres has been assumed by Flechsig to pass continuously through the red nucleus of the tegmentum and thalamus into the internal capsule, to be distributed along with the fibres of the pyramidal tract to the region of the **Rolandic fissure.** By this means a crossed connection would be established between the **central gyri** and the **cerebellum.**

(*e*) Fibres from the **olfactory bulb,** after decussation in the anterior commissure, are also believed by Meynert to join the optic radiations of Gratiolet, to be distributed to the occipital and temporo-sphenoidal cortex.

We have seen how those peduncular fibres forming the crusta diverge and enter the hemisphere as the internal capsule, supporting upon its upper surface the caudate nucleus and thalamus, and receiving, into its concavity below, the wedge-shaped mass of the lenticular nucleus, whose apex contributes a mass of fibres to the formation of the crusta. If we now imagine a lamella of fibres spread over the broad base or outer aspect of this wedge to pass downwards beneath the lenticular nucleus towards its apex, and there meeting the crusta, whose fibres it crosses transversely to its inner side, we have an arrangement which, from the insula and temporal regions of the brain, passes to the base, as a sort of sling-like loop supporting these ganglionic masses, and binding them and the peduncles together.

Such an arrangement is represented by the external capsule and its continuation at the base, where it is termed the **ansa peduncularis** of Gratiolet or **substantia innominata** of Reil; and a portion of which crossing the crusta to its inner side is named the **collar, fillet,** or **loop** of the **crus.**

This important formation of the ansa peduncularis consists of four systems of fibres, according to the statement of Meynert. They arise from the under surface of the lenticular nucleus, from a ganglion lying in this position, and from the cortex of the sylvian fissure, upper and temporal margin of the insula. The capsular portion of the ansa has no organic connection with the base of the lenticular wedge over which it spreads, so that it can be readily separated, and the latter enucleated by the handle of the scalpel. Its fibres, which arise from the cortex of the insula and upper margin of the sylvian fissure, necessarily pass to it through the **claustral formation**; and then, forming the compact lamella of the external capsule, converge to the fasciculi at the base of the lenticular nucleus, which we referred to as the ansa. At this point it is crossed superficially by the anterior commissure, which has to be removed to expose it in its entirety. The deepest layer of the ansa peduncularis takes its origin from fibres issuing at the base of the lenticular nucleus —from those concentric lamellæ, the **laminæ medullares.** The fasciculus completely crosses the crusta parallel to, and immediately in front of, the optic tracts, and passing to the inner or median border of the crusta, forms the innermost series of fibres, here destined to pass back along the **raphé** to the central grey substance, where they terminate after decussation in the **nucleus of origin** of the oculo-motor and trochlear nerves within the **nates.** The second layer originating in fibres from the cortex of the upper margin of the sylvian fissure, the temporal lobe and the cortex of the insula, is interrupted in an elongated ganglion at the base of the lenticular nucleus. From this ganglion fresh fibres proceed at first parallel to the course of the rest of the ansa, but then suddenly bending backwards and upwards, pursue their further course just within the grey substance of the third ventricle, forming the well-marked bundle of medullated fibres known as the **posterior longitudinal fasciculus,** which may possibly be traced down into the spinal cord as the most posterior of the fibres of the anterior column in front of the grey commissure of the cord.

The third layer of the ansa arises from the **sylvian fossa** and the cortex of the temporal lobe, runs parallel to the above-mentioned fasciculi at the base of the lenticular nucleus; and then turning upwards into the thalamus, forms a brush-like radiation of its fibres far back into the interior, constituting the so-called **inferior peduncle** of the thalamus. A fourth layer of the ansa which overlies the latter joins the **stratum zonale,** or capsular investment of the thalamus. This substantial belt of the ansa firmly binds together the region of the **operculum** and **island** to the central structures at the base of the brain, forming a complete sling around the lenticular wedge, consisting of a series of loops, the deepest of which connects the base of the lenticular body to the motor nuclei in front of the aqueduct; the others having a more lengthened course from the cortex to the anterior column of the spinal cord, or blending intimately with the structures of the optic thalamus and its capsular investment.

Dissection.—Place the brain with the base uppermost, having carefully removed the membranes and the large vessels. Remove with the blade a shallow horizontal slice from one of the temporo-sphenoidal lobes so as to pass through the hook of the **uncinate gyrus;** this lays bare the section of the **pes hippocampi,** and the **amygdaloid nucleus.** Pass the handle of the scalpel vertically through the medullary strands outside the pes—it enters the extremity of the descending horn of the lateral ventricle. Carry an incision along the floor in a somewhat curved direction, backwards and outwards, along the whole length of the occipito-temporal gyri. This exposes the descending cornu throughout its extent, and the worm-like **cornu ammonis** is seen descending from above to terminate at the pes. The **tails** of the **caudate** and **lenticular nuclei** are now seen along the *roof* of the cornu, and a white, glistening, narrow band of fibres curves downwards towards the **amygdala**—the **tænia semicircularis.** Upon raising up the inner border of the uncinate gyrus, we expose the optic tracts arching backwards over the crusta, and terminating in the two elevations of the corpora geniculata, externa and interna, beyond which the **pulvinar** of the thalamus projects. In front of the optic tracts the crusta has disappeared in the depths of the hemisphere, and we see here the triangular floor of the anterior perforated space bounded by the delicate white roots of the **olfactory** nerves. Drawing the optic nerves backwards we expose the delicate, almost translucent grey lamina forming the floor of the fifth ventricle, bounded by the two white peduncles of the **corpus callosum,** which at this juncture probably tears across, revealing a broad white fasciculus, the **anterior commissure,** crossing from one to the other hemisphere.

Upon gently drawing apart the divergent crura cerebri in front, a dark line is apparent, running the whole length of its mesial aspect at a greater depth than the innermost fasciculi of the crusta. This, upon dividing the crura, is seen to be the inner margin of a dark pigmented body, of lenticular section, the **substantia nigra** of Soemmering, which forms a distinct boundary wall betwixt the crusta in front and the tegmentum behind: it extends from the level of the corpora albicantia as far as the upper border of the pons. We shall refer more minutely to its relationships when studying transverse sections of this region. Immediately behind lie the tegmentum and the large ganglia of the mid-brain or quadrigeminal bodies.

We can best study this posterior division of the mesencephalon by first examining the external configuration and connections of these ganglia, and subsequently following the course of their fibres, and next by studying the relative position of the fasciculi as seen in transverse section at different levels. If now we so separate the occipital lobes of the hemispheres as to expose these bodies to view from above, they appear in a mid position between the two divergent cushions of the thalamus, overriding the cerebral peduncles in the form of two pairs of tubercles—an upper and a lower, each oval in form, the upper pair the larger and darker in colour, both having their long axis disposed transversely. Both pairs of tubercles extend upwards and forwards an arm, which is really a connection for the cerebral cortex, and spreads upwards as **coronal radiations**: each pair in like manner sends downwards a medullated lamella on either side of the crus cerebri—that from the nates being deeper-placed and over-laid by that from the testes; this downward extension is called the **fillet** of the quadrigeminal bodies, or the **lemniscus.** We shall trace the arms or **brachia** and the **lemniscus** at a subsequent stage of our examination.

Between the nates, at the upper end of their median raphé, lies the **conarium** or **pineal body** (fig. 4), with its long delicate **brachia** extending forwards, and bounding the white ventricular surface of the thalamus above, from the median grey walls of the third ventricle. Beneath and in front of the pineal body is the **posterior commissure,** under which we observe the opening of the **aqueductus sylvii** into the third ventricle. Hence the quadrigeminal bodies lie immediately over the continuation of the central grey substance of the ventricles surrounding the aqueduct, which would be, therefore, laid open by a vertical incision carried through the median raphé of the quadrigeminal bodies. Moreover, the descending ribands called the upper and lower lemniscus are, in their descent, closely approximated to the outer wall of the central grey substance, which is, therefore, as it were, very largely embraced by the quadrigeminal system.

Just external to the central grey substance are the various structures entering into the formation of the tegmentum, extending as far forwards as the substantia nigra. If the peduncular fibres of the crusta exposed on one side of the pons, upon removal of the superficial layers of the middle peduncle, be divided, raised, and the deeper transverse fibres be dealt with in like manner, we come down upon the most anterior layers of the tegmentum, and these, when traced, are found to be the fibres of the lemniscus, which winds round from behind, and insinuating itself beneath the overlapping crusta, passes in a compact belt extending even as far as the median raphé of the pons (fig. 5, F). In its further course it is traced downwards as the most external zone of the lateral columns of the spinal cord in front of the **direct cerebellar column.** Whilst the lemniscus in this part of its course gradually encloses the several other tegmental structures, including the red nucleus, the fibres given off from the latter after decussating at the median raphé pass to the outer side of the tegment in its further course, and eventually escape from the embrace of the lemniscus about the level of the upper transverse fibres of the pons, and, becoming superficial, pass downwards to the cerebellum as its superior peduncles. We must now follow the fasciculi of the **mesencephalon** in transverse sections taken across the upper and lower pairs of quadrigeminal bodies.

In such sections passing through the region of the **nates,** five structures which demand examination are exposed to view; these are in order from behind forwards—(1) The nates; (2) the central grey substance; (3) the tegmentum; (4) the substantia nigra; (5) the crusta.

(1) **The Nates** (Superior Bigeminal Body).—The ganglionic structure presents four distinct strata—(*a*) Outer grey or cortical layer of Forel. (*b*) Longitudinal fasciculi from the external geniculate body. (*c*) Bundles of fasciculi passing outwards on all sides from the central grey substance in delicate radiations into the substance of the ganglion. (*d*) The stratum of the lemniscus, embracing the grey substance, and through which the radiating fibres last named have to pass outwards. To appreciate correctly the structure of these ganglia, we must imagine a coronal fan of fibres from the cerebral cortex converging in the direction of the external geniculate bodies into the substance of the cortical layer of the nates; passing back to the median raphé behind the aqueduct, and decussating here with a similar fasciculus from the opposite hemisphere; thence arching around the central grey as the lemniscus already described. The coronal fasciculus constitutes the **superior brachium** of the nates; and, *prior to the decussation,* it terminates in the ganglionic cells of the nates. From these cells arise the fibres which decussate, and pass as the lemniscus downwards into the spinal cord. We have, therefore, the two ganglia of the nates, so to speak, enclosed between the upper and lower arms of a decussating medulla with an internode of ganglionic cells on the centric side of the decussation; and the lower arms or **stratum lemnisci** enclosing in like manner the central grey, and supporting as a girdle the mass of tegmentum lying in front of the latter. The upper arm or brachium passes in part direct to the cortex beneath the pulvinar of the thalamus, and thence through the posterior division of the internal capsule; and in part, passes into the external geniculate body behind and covering the former. This decussating system of

medulla extends downwards, presenting a similar formation for the testes as for the upper bigeminal body (nates). Here also we have an **inferior brachium** of the testes, and an **inferior lemniscus** given off to the medulla and cord. As we shall see later on, an almost exact counterpart of this medullary system carried forwards explains the formation of the posterior commissure of the third ventricle.

Both geniculate bodies receive coronal radiations, both transmit fibres through the brachia to the corresponding quadrigeminal body, and the external geniculate is intimately connected with the optic tract as the latter passes to the nates by the medium of the upper brachium; whilst the inferior brachium and hence the testes have likewise a connection with the inner side of the tract. Hence the nates and testes are brought into relationship with the cortex of the occipital and temporal lobes, as well as with the retina. The radiating fibres spreading from the central grey substance in the nates through the lemniscus, are probably direct connections between the nerve cells of the nates and the nuclei of the oculo-motor nerves within the central grey area. In the region of the nates also we observe in cross-sections numerous fasciculi lying between the antero-lateral margin of the grey substance and the lemniscus; they pass inwards between the red nucleus and the posterior longitudinal fasciculus and decussate at the raphé—their destination being obscure. By Meynert they are supposed to be connected with the nucleus of the descending root of the fifth nerve.

Red Nucleus of Tegmentum and Upper Cerebellar Peduncle.—When describing the structure of the thalamus we shall find, as an important constituent, a rounded nuclear mass named the **red nucleus,** which, upon the one hand, receives coronal radiations, and, on the other hand, gives off medullary fasciculi extending downwards through the quadrigeminal region, where they decussate at the median raphé to terminate as the superior peduncles of the cerebellum. The nucleus itself, consisting of much grey matter enclosing large and small nerve cells, is continued into the region of the nates, below which its grey matter disappears, and white medullary fasciculi with interspersed nerve cells are alone continued downwards to the points of decussation. In the upper sectional planes (transverse) of the mesencephalon, we see this red nucleus of almost rounded contour embraced between the substantia nigra and commencing lemniscus (in front and laterally) and the other fasciculi of the tegment behind. At this level also the arched roots of origin of the oculo-motor nerve lie on its inner side, and partly traverse its structure.

In the lower planes of the mid-brain, through the testes, the medullary fasciculi derived from the red nucleus, now called the

superior cerebellar peduncles, approach the median raphé and decussate completely with the fasciculi of the opposite side. Throughout this decussation the fasciculi are embraced between the loop of the lemniscus in front, and the posterior longitudinal fasciculi behind. From the line of decussation the fasciculi now arch outwards and backwards, and, still covered by the lowest fibres of the lemniscus derived from the testes, emerge opposite the greatest convexity of the pons to enter the cerebellum upon the same side. If we adopt Meynert's view of the **projection system,** the **nucleus ruber** forms an **internode** or point of interruption between the coronal fibres of the cerebral hemisphere and the superior peduncular fibres of the opposite cerebellar hemisphere; and, as with the first link of the projection system generally, *the internode occurs on the same side* as the hemispheres supplying the coronal attachments.

Posterior Longitudinal Fasciculus.—A compact column of large nerve fibres, oval, somewhat pyriform or lenticular, according to the plane of section, presents itself immediately in front of the central grey area, and, therefore, *behind* the red nucleus or its decussating medulla—the **cerebellar peduncles.** This very obvious column of fibres is seen as one of the most striking features of the tegmentum throughout the mesencephalon and down the whole of the medulla oblongata. We have already seen that this fasciculus originates in a compressed ganglionic mass forming the second stratum of the ansa peduncularis, and that its *coronal* origin is from the cortex of the operculum, insula, and temporal lobe; it is traced into the posterior fibres of the anterior columns of the spinal cord.

Substantia Nigra of Soemmering.—Another formation seen in these transverse sections is the grey matter of Soemmering. It begins near the posterior plane of the corpora albicantia, and stretches downwards to the lowest limits of the mesencephalon, terminating, therefore, where the transverse fibres of the pons appear. For the greater part of its course it stretches completely across the mesencephalon in an oblique direction forwards, a line which, if continued, would meet that of its fellow at an acute angle. It owes its dark colour to an abundance of large pigment cells. We shall, when referring to the thalamus, find that in transverse vertical sections through the hindmost part of the third ventricle, this grey matter lies between the crusta and the red nucleus, and that a fan of coronal fibres is here seen passing outwards beneath the thalamus to the cortex (*Meynert*). Fibres pass downwards and forwards from this grey belt into the middle and inner divisions of the crusta, and hence this substance forms a ganglion of origin for certain portions of the crusta of the cerebral peduncles, and although resting close upon the tegment behind, has no organic connection therewith.

We have thus traced the several ganglionic structures and medullary fasciculi, entering into the formation of the mesencephalon, and it remains but to summarise the results of the inquiry. The mid-brain consists of two pairs of ganglia, the quadrigeminal bodies seated upon the **brain stem** or **pedunculus cerebri,** where it diverges as two branches or crura, uncovered by the transverse layers of the pons, and up to the point of its entrance into the base of the brain. The cerebral peduncle consists of **crusta** and **tegment** severed by the intervening **substantia nigra.** The nates and testes have intimate connections through their *brachia* with the cerebral cortex and retina; and below through the girdle-like lemniscus with the olivary bodies, and, according to Meynert, the lateral columns of the cord; both ganglia are also connected by their radiating central fibres with the oculo-motor nuclei in the central grey substance continued from the ventricle. In front of the ganglia and central grey substance lies the structure of the tegment, viz.:—The posterior longitudinal fasciculus; the superior cerebellar peduncular fibres and its red nucleus of origin; certain fasciculi crossing the median raphé from the quadrigeminal bodies; and lastly, the layer of the lemniscus. Anterior and external to the tegment, is the crusta with the substantia nigra lying behind it. In the crusta we recognise the pyramidal tract as occupying the inner, middle third, and the portion behind and between these areas, representing respectively the accessory, fundamental, and mixed tracts; whilst in the outer fourth pass the fasciculi of the sensory tract. In high planes and upon the innermost fibres, the deepest layer of the ansa peduncularis passes backwards to the nucleus of the oculo-motor. Lastly, the substantia nigra, peculiar to this region, represents a ganglion, from which the crusta in part arises, and which in itself is but an internode for coronal radiations.

THE THALAMENCEPHALON.

The region of the thalamencephalon is best exposed within the lateral ventricles, for the study of its superficial parts and their relations; and, for this purpose, a dissection, such as shown in fig. 7, should be made, in which the relative position of the mesencephalon and epencephalon are equally exposed. We here see the two great ganglia, the optic thalami, the pineal gland with its peduncle, the central grey substance (and the commissure) of the third ventricle passing downwards towards the infundibulum, and the two corpora geniculata beneath the hinder extremity of the thalamus indicating the termination of the optic tracts. These, then, form the chief structures constituting the **"tween-brain"** or **thalamencephalon.** To appreciate their mutual relationships—their centric and peripheric connections—a careful study of sections carried through this region in three different planes is requisite—viz., horizontal, longitudinal, and vertical-transverse; but a preliminary study of their more obvious external conformation is necessary ere a more minute inquiry is instituted. The reader should refer here to the illustration (fig. 7) given on p. 30.

The optic thalami are somewhat oval, wedge-shaped bodies, broadest behind, where they diverge from each other so as to expose the quadrigeminal bodies; and narrowed anteriorly where they approach the middle line. They are limited externally by the **stria terminalis** (cornea), which is also the upper and outer boundary of the thalamencephalon—immediately external to which is the caudate nucleus and its attenuated tail. In front, the thalamus presents a notable prominence, the **anterior tubercle;** behind, it projects back as the **pulvinar,** and forms in the descending horn of the lateral ventricle the anterior wall or roof of the cornu. Mesially, the thalami are bounded by the **peduncles** of the **pineal body;** and the vertical median grey walls of the third ventricle do not, as might be conjectured, represent the median aspect of the thalami, but must be carefully distinguished therefrom. In fact, the mesial aspect of these grey masses is here completely concealed beneath the grey matter of the third ventricle, which is identical and continuous with the central grey substance of the cerebro-spinal tube throughout its length. To the thalami, however, belong the middle and posterior commissures which cross the ventricle, and which are really **decussating medullated tracts** of the thalami.

Whilst the inner face of the thalami covered by the central grey substance is perpendicular, the outer presents a kind of obliquely sloping roof resting upon the fasciculi of the internal capsule; and hence the vertical transverse section of the thalamus is likewise somewhat wedge-shaped in configuration. Then again, it must be remembered that the thalami in lower vertebrates—birds and reptiles—are very evident projections on the upper surface of the peduncles not included within the hemisphere at all; and that in man, although they appear thus to project within the ventricles and to be included within the more extended hemisphere, they, in reality, are *outside* the hemisphere of which the fornix constitutes the median boundary.

We have spoken of the thalamus as a somewhat wedge-shaped mass, as seen in transverse vertical sections. In similar sections through both thalami, they conjointly appear like the transverse-section of a boat, keel downwards, in which the arched side rests as on a couch in the concavity of the internal capsule, whilst in the hollow of the keel the thalamus is separated from the capsule by a region known as the sub-thalamic region, in which a sharply-defined, biconvex, lens-shaped body is situated, to which we shall refer later on as the

sub-thalamic body (lenticular body of Meynert, or Luys' body of Forel). The anterior end of the thalamus, therefore, is placed at a considerable distance from the base of the brain and the sub-thalamic region—the mass of the cerebral peduncles and the intervening substantia nigra being immediately beneath it; whilst the whole extent of the central cavities of the third ventricle and its grey walls continuous below with the infundibulum and posterior perforated space, must be excluded from the true thalamic structure.

The interior of the thalamus consists of a large mass of grey matter, split into layers in various directions by the medullated strands passing into its structure. The grey matter encloses numerous nerve cells, which are the thalamic termini for coronal radiations connecting the most diverse regions of the cortex with this body, and the centres of origin for fresh strands which pass down into the *tegmentum* of the crus. The arrangement of medullated and grey elements is peculiar. The cortical fasciculi, as they enter the thalamus, diverge within its structure in brush-like fashion, forming concentric lamellæ, between which are intercalated the layers of grey matter with their nerve cells. Since medullated fasciculi enter the thalamus from very distant regions of the cortex—from the frontal, occipital, temporo-parietal gyri and gyrus fornicatus—they necessarily meet at varied angles, and cross each other in their course within the ganglion: thus it is that this body becomes moulded by its medullary cones into apparently distinct segments—not, however, true centres or nuclei in the usual acceptation of the term, since their grey substance freely intermingles with that of neighbouring structures.

The cortical contribution to the medullated system of the thalamus approaches that body in part at its anterior extremity by three so-called **peduncles**—the anterior, superior, and internal (or inferior). These thalamic peduncles connect its structure with the frontal lobe, the sylvian fossa, temporal lobe, and gyrus fornicatus respectively. Such cortical fasciculi have necessarily a lengthened course to pursue, and none more so than that from the **gyrus fornicatus,** which reaches its destination after a peculiarly complex spiral course. The anterior peduncle approaches the thalamus from the frontal cortex through the strands of the *anterior segment* of the internal capsule between lenticular and caudate nuclei, interlacing here with its fibres, and eventually passing into the front end of the thalamus, expands brush-like in its interior, its fibres arching backwards, crossing the fibres of the inferior peduncle, and passing chiefly to the outside of the latter. A portion decussates at the middle and posterior commissure, whilst the rest continues directly down the tegmentum. Part of its fibres help in the formation of the capsule of the thalamus or so-called **stratum zonale.** The **inferior**

peduncle, already alluded to as the third layer of the ansa peduncularis, connected with the cortex of the temporal lobe, passes from beneath the lenticular nucleus up into the thalamus, expanding also in brush-like manner chiefly along its internal portion, and forming the inner boundary of the thalamus. It also decussates at the middle and posterior commissure to pass down as *tegmental* fasciculi of the crus.

Both these peduncular expansions are interrupted by the nerve cells in the grey intercalated layers of the thalamus ere they decussate at the commissures. The **superior peduncle** takes a still more complicated course; its centric connection is with the cortex of the gyrus fornicatus—appearing first in the two **fimbriæ** or **posterior pillars** of the **fornix** arising from the cornu Ammonis; and ascending as the body of the fornix connected by the transverse fibres of the **lyra** upon the thalamus, it arches forwards at the front end of this body, and thence passes downwards as the two **descending pillars** of the fornix. These latter pass back to the corpora albicantia, around which they form a distinct loop, and again turn upwards as the **ascending pillars** or bundles of Vicq D'Azyr to terminate within and around the anterior tubercle of the thalamus. Whilst forming this loop around the **corpus albicans,** a portion of its fibres is interrupted by nerve cells within this body, and a fasciculus starts from this site and passes directly backwards into the tegmentum.

Whilst the frontal, insular, and median cortex is thus connected by the thalamic peduncles to the anterior end of the ganglia, the posterior or hinder half of the thalamus receives along its outer margin coronal radiations from the occipital and mid-regions of the hemisphere. These fasciculi radiate from the upper and outer border of the thalamus to corresponding regions of the brain opposite them; the middle section spreading towards the mid-regions; and the posterior arching backwards towards the occipital pole. These latter, as they pass outwards and backwards to the occiput, are associated with similar radiations proceeding in like direction from the geniculate bodies and the brachia of the nates and testes. This system of fibres arches around the outer wall of the posterior cornu of the ventricle, and has long been known as the optic radiations of Gratiolet. In their course they are brought into close association with the sensory fibres of the cord destined for the occipital and temporal lobes; and, as we have previously seen (fig. 7, **S**), occupying the outer fourth of the crusta. This peduncular sensory tract, it must be remembered, has no connection with the optic thalamus, but runs directly into the occipital and temporal regions of the cortex. The coronal radiations which enter this outer border of the thalamus, pass through its structure as *arched fasciculi* towards the median line—*i.e.*, across the long axis of the thalamus; the medullated tracts being intercalated by the grey matter common to the whole ganglion. Upon a lower level than the entrance of these cortical radiations, other medullated fasciculi pass into its substance in an identical manner from the middle root of the optic tract, and this double origin partially severs this hinder region of the thalamus into an upper and a lower segment. In *both systems* of fibres, hemispheric and retinal (through the optic tract), union of the fibres is effected with the cells of the grey intercalated layers.

It has been shown that the peduncles—anterior and inferior—entering the anterior pole of the thalamus, run backwards through its structure as brush-like formations to terminate in cells of the grey matter ere they decussate at the commissures; and that a larger proportion of these fresh fasciculi *do not decussate*, but pass *directly* downwards into the tegmentum. The latter *direct fasciculi*, in passing into the hinder half of the tegmentum, run immediately across the axis of the **optic** and **cortical radiations** just described; and necessarily form apparent concentric dissepiments in these regions. These laminated dissepiments form the new medullated tracts for the tegmentum arising within the grey matter of the thalamus. The anterior peduncle especially, passing backwards through the thalamus, is not crossed by these transverse radiations, and its region is bounded on the outer side by a strongly-marked medullated belt, the innermost of the concentric dissepiments alluded to, and known as Burdach's **lamina medullaris.** This well-marked boundary and absence of transverse radiating fibres, maps out a kind of nucleus in this region of the anterior peduncle, which is known as the **centre median** of Luys.

On examining the thalamus from above, after opening-up the lateral ventricles, it is found that the grey matter forming the tail of the caudate nucleus may, by gentle pressure with a brush, be raised away, together with the stria cornea, from the subjacent parts; and, immediately beneath it, radiating fibres in coarse fasciculi are seen passing from the whole extent of the upper margin of the thalamus, either directly outwards towards the parietal lobe, or arching back towards the occipital region. These fasciculi consequently form the outer wall of the lateral ventricle in their course towards the parietal lobe. If the scalpel divide these fibres across parallel to the direction of the stria cornea, the blade passes directly into the internal capsule, and it becomes evident that the outer obliquely-placed surface of the thalamus rests upon the internal capsule as upon a couch, and gives off from the whole of its outer aspect medullated fibres which enter into the constitution of this capsule, and then spread as coronal radiations to the various districts of the cortex of the parietal and temporo-sphenoidal lobes. The greater bulk of these pass *deeply* into the thalamus, and, as before said (p. 43), are crossed by the brush-like fasciculi of the thalamic peduncles. The more superficial layer first revealed upon raising the tail of the caudate, enters into the constitution of the **white capsular investment** of the thalamus (*stratum zonale*), which gives to this ganglion its peculiar white hue within the ventricle, as contrasted with the greyish aspect of the caudate nucleus.

The capsule of the thalamus spreads inwards as far as the peduncle of the pineal gland ere it turns downwards to form part of the inner investment of the thalamus; and at this line it disappears from view, and the grey matter of the third ventricle becomes apparent. This capsule or stratum zonale is itself of complex formation: it receives also fibres from the optic tracts, the uppermost of those which join the thalamus; so also fasciculi from the frontal lobe enter it by the anterior peduncle of the thalamus, and in like manner the most superficial stratum of the *ansa lenticularis*; lastly, the gyrus fornicatus sends its contribution by means of the ascending pillar of the fornix, which in this course embraces a nodular segmented portion of the thalamus at its anterior extremity, termed the **anterior tubercle.** Hence the zonular layer or thalamic capsule receives fibres from almost every region of the brain—the frontal, parietal, temporo-sphenoidal, and occipital lobes, and the mesial aspect or gyrus fornicatus, as well as the retina. This very extensive retinal and hemispheric connection of the thalamus may be thus tabulated :—

Fasciculi from	
Frontal lobe, ...	. Through anterior peduncle of thalamus.
Temporo-sphenoidal lobe,	. Coronal radiations and superficial layer of ansa lenticularis.
Parietal and occipital lobes,	. Coronal radiations along its whole outer surface.
Gyrus fornicatus, . . .	. Through pillars of fornix.
Retina, ...	. Through uppermost thalamic connections of optic tract.

The Pineal Body and its Connections.—Surrounding the upper pair of the quadrigeminal bodies, immediately beneath the posterior extremity of the callosal commissure and in the middle line between the mesencephalon and diencephalon, lies a small, reddish, somewhat conical structure—the **pineal body.** It is closely attached to the **velum interpositum,** so that it is frequently torn away with the membranes investing it. It is hollowed into several small sacculi, which contain the gritty, earthy, and amylaceous material termed **acervulus cerebri:** and the structure is peculiarly vascular. In microscopic structure we find it consists, like other ganglionic structures in the brain, of closely aggregated cells, varying considerably in size from 5 μ to 18 μ.

Its connection with the rest of the cerebrum is effected by means of two processes, which are directed forwards along the inner border of the thalami optici, forming a boundary between the latter and the grey matter of the third ventricle; and descending in front in conjunction with the tænia semicircularis and the pillar of the fornix: these are the two **peduncles** of the pineal body or **habenula.** These peduncles are distinctly ganglionic in structure, and together with the pineal body are probably to be regarded, as Meynert believes, as *ganglia of origin* for the *tegmentum.*

The connections by medullated tracts are twofold—centric and peripheric. The former, as a connection with the cerebral hemispheres, takes place through the medium of the *stratum zonale*, already described as investing the *optic thalamus.*

The latter or peripheric connection is effected by a large and important fasciculus, which passes down vertically from the habenula or peduncle, covered by the grey matter of the third ventricle, and towards the region (at the base) of emergence of the motor oculi nerve on the inner side of the converging *crura.* In this course it describes a sigmoid bend, and near the base of the mesencephalon it lies between the posterior longitudinal fasciculus, on the median aspect, and the *red nucleus* of the *tegmentum*, external to it. Some of its fibres radiate into the *nucleus ruber* (Meynert); but the larger proportion bend at this point immediately backwards at right angles to their former course, and appear to pass into the tegmental areas of the pons and medulla, in conjunction with the *posterior longitudinal fasciculus.* This rectangular bend has gained for it the appellation of the **fasciculus retroflexus**—it is often termed the **style** of the **peduncle** of the pineal body, where it passes vertically towards the red nucleus of the tegmentum.

The *style* or *fasciculus retroflexus* may be best exposed by transverse vertical sections carried through the ganglion of the peduncle just in front of the quadrigeminal bodies, but it may also be traced in longitudinal vertical sections near the mesial plane of the "*tween* and *mid* brain." In these sectional planes, however, owing to its sigmoid flexure, a part only of its course can be usually seen. Thus in a vertical longitudinal section of the brain of the dog, near the mesial plane, we find the lower end of this fasciculus about to bend backwards at right angles, and on this plane it is seen to descend in front of medullated fasciculi passing downwards from the posterior commissure and the emergent roots of the third nerve. In a section carried still nearer the mesial plane, we see its course about complete, whilst a portion of both ascending and descending pillars of the fornix is revealed likewise.

Posterior Commissure.—We have already traced the anterior and inferior peduncles of the *thalamus* as far as their decussation in the posterior commissure, and it would seem extremely probable that the *fasciculus retroflexus* undergoes partial decussation through the medium of this commissure also. Near the mesial line, we can readily trace these decussating fibres of the posterior commissure in their further course passing downwards into the tegmentum, where they bend backwards to pass into the medulla and spinal cord; whilst prolonged from the posterior commissure backwards is also seen the cross-section of the medullated fibres of the corpora quadrigemina.

In these vertical longitudinal sections taken near the mesial plane, we therefore see three systems of decussating fasciculi crossing at the middle line, and forming peripheric extensions from a series of ganglionic bodies, viz.:—The **fasciculus retroflexus,** the mass of the **posterior commissure,** and the quadrigeminal fasciculi called the **lemnisci** or **fillets** of the **nates** and **testes.**

Corpora Geniculata.—Beneath the pulvinar of the thalamus in man we see a small club-shaped body about the size of a coffee-bean, directly continuous with the optic tract anteriorly, and by a notable border separating *mesencephalon* from *thalamencephalon*, connected with the upper quadrigeminal body or *nates*. This small ganglion, for ganglionic it is in nature, is the **outer geniculate body,** and lies in the course of the arm of the nates or superior *brachium*, with which it is intimately connected, as it proceeds to the cortex of the occipital lobe. Upon vertical longitudinal section it is found to possess a peculiar plicated arrangement of a medullated and a grey lamina, exhibiting alternating layers of grey and white substance.

Internal to this body, that is, nearer the mesial plane, lies another small structure of spindle-shaped outline, immediately beneath the upper and between it and the lower brachium; it is directed towards the nates by one of its pointed extremities. This structure is the **inner geniculate body.** Both geniculate bodies are connected with the corpora quadrigemina on the one hand, and with the cortex of the occipital lobe along with the other centric fasciculi of the brachia. In a vertical section we find an extensive portion of the optic tract directly continuous with this plicated outer geniculate ganglion, and hence also with the nates. An inner segment of the optic tract, but much more limited in extent, passes into the internal geniculate and thence to the *nates* also; no fibres from the optic nerve are believed to pass by this tract to the *testes*.

The remaining connections of the optic tract are the optic thalamus (to the *stratum zonale* and radiating fasciculi previously described); and the basal optic ganglion, a small body of grey matter lying beside the *tuber cinereum* immediately covered by the optic commissure.

The ganglia of origin of the optic nerves, therefore, are the upper quadrigeminal, the outer and inner geniculate bodies, the optic thalamus, and the basal optic ganglion: the centric or coronal extensions arising in these ganglia pass by means of the posterior section of the internal capsule as the optic radiations of Gratiolet to the cortex of the occipital and (?) temporo-sphenoidal lobes.

PROSENCEPHALON OR FORE-BRAIN.

Configuration.—We have already seen that divergence of the brain-stem in the crura cerebri to reach either hemisphere, entails also the more and more complete severance of the various ganglionic masses at the base with which it is brought into connection: and that from the bilateral fusion of the mesencephalon, we pass forward to the divergent masses of the thalami (diencephalon), and thence to the still further severed corpora striata, constituting the ganglia of the fore-brain (prosencephalon). We have seen how these more divergent masses are braced together by sling-like loops of medulla, such as the ansa lenticularis, and united mesially by the anterior and other commissural tracts. The ganglia of the prosencephalon form the most anterior mass of grey matter surrounding the peduncular extensions, and are so disposed as to constitute two incompletely-severed masses of grey substance, whose configuration shadows forth the form assumed by the hemispheric envelope moulded around them. The flexure of the fore-brain, whereby this hemispheric arc reproduces the contour of these ganglionic structures, has its site at the fissure of Sylvius; and, in fœtal brains, ere the further differentiation of the cortex into its varied longitudinal and transverse fissures has proceeded, we see readily how the hemispheres are, so to speak, moulded after the form of their subjacent ganglia. The axis of this flexure is constituted by the most external of these ganglionic masses, the so-called **lenticular nucleus,** wedge-shaped in form, its base directed forwards and outwards, covered by the cortex of the insula—its apex downwards and inwards towards the crus cerebri. Around this wedge-shaped axis, the ganglionic and hemispheric arcs are severally formed—the ganglionic, in the form of the caudate nucleus: the hemispheric, beginning at the orbital aspect of the frontal, sweeps round the fronto-parietal to the tip of the temporo-sphenoidal lobe. The more flattened aspect of the region of the insula, therefore, bears the impress of the base of this **lenticular** axis of revolution, whilst the more spheroidal contour of the hemisphere conforms to the curvature of the **caudate** body.

Upon this constructive principle largely depends the divergence observed in the primitive contour of the cerebrum in various animals and in man. Although identical in the nature of their histological constituents, these two ganglionic masses differ widely, not alone in their rough contour, but in their quantitative relationships: in certain brains, the caudate nucleus assumes a mass far out of all proportion to the lenticular; whilst in man, the former is dwarfed, and the latter assumes a relatively important *rôle*. The greater magnitude assumed by this lenticular axis of revolution, the greater the scope for the unfolding of the hemispheric arc, and the more important the developmental features assumed by the regions of the insula and sylvian fossa. On the other hand, the smaller lenticular body, and the larger proportionate development of the caudate bespeak a brain of simple configuration, more spherical, less complex in convolutionary arrangement, and of more uniform symmetry throughout.

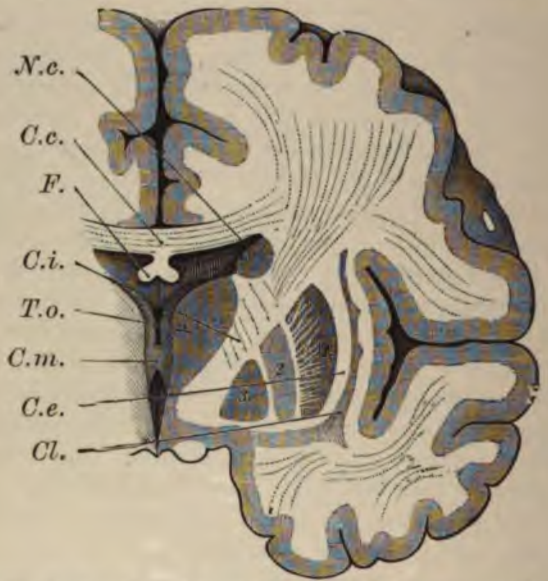

Fig. 8.—Section through hemispheres (vertical transverse) through plane of middle commissure.

N.c, Tail of caudate nucleus.
C.c, Corpus callosum.
F, Fornix and choroid plexus.
C.i, Internal capsule.
T.o, Optic thalamus.
C.m, Middle commissure.
C.e, External capsule.
Cl, Claustrum.
1, *2*, *3*, Three segments of lenticular nucleus.

In thus indicating their impress in the configuration of the cerebral vault, these striate ganglia differ widely from the **diencephalic ganglia** previously considered, the **thalami optici**; in fact, these latter bodies, so far from having any portion of the cerebral hemisphere moulded to their form, are themselves wholly *outside* the cerebral envelope in their mesial position. Thus, the adult brain witnesses to the genetic relationship of the cerebral hemispheres, and the related striate ganglia; the whole mass in front constituting the **fore-brain** or **prosencephalon** in advance of the **thalamencephalon.**

We have spoken of these basal ganglia as incompletely severed masses of grey matter, a statement at once verified by vertical sections taken in anterior planes through these bodies. In such anterior planes, the medullated interval elsewhere separating these bodies is bridged by numerous broad bands of grey substance which are but extensions from one to the other ganglion; whilst, at the base, complete fusion occurs between the two, the head of the caudate nucleus merging into the frontal extension of the lenticular, becoming so superficial at the base as to be merely covered over by the orbital medulla and the grey matter of the anterior perforated space with which it becomes continuous.

Caudate Nucleus.—The innermost or intraventricular nucleus of the ganglion of the fore-brain, and the only portion superficially seen within the lateral ventricles, is of pyriform shape, with a long attenuated tail-like process extending into the temporal lobe. In this course, as before explained, it is bent upon itself, its axis of revolution being the lenticular body. The head of the ganglion fusing at the base with the lenticular, arches forwards and inwards towards the **septum lucidum,** and, lying on the inner aspect of the internal capsule, embraces, in this first part of its course, an important medullated fasciculus, which connects the cortex of the frontal lobe with the anterior extremity of the optic thalamus, the so-called **anterior thalamic peduncle.** In its further course, it ascends above the level of the lenticular, and lies upon the internal capsule; its tail-like extension resting, opposite the thalamus, upon the hemispheric fibres which pass beneath it, to form the capsular investment of the thalamus (*stratum zonale*). Still further back, the tail arches downwards into the descending horn of the ventricle, and can be traced upon the roof of the latter as far forwards as its anterior extremity, where it terminates in a somewhat bulbous end, having immediately in front of it a mass of grey matter, termed the **amygdala.** It will be seen from this description that the bulbous extremity of the tail extends almost as far forwards as the head of the caudate nucleus, and thus describes an almost complete loop around the internal capsule and thalamus, hence termed the "**surcingle.**" The whole course of this loop can be well demonstrated by vertical longitudinal sections of the hemisphere; whilst vertical transverse sections anywhere between the amygdala and posterior end of thalamus reveal the upper and lower segments of the surcingle, as isolated grey masses above and below the thalamus. Each of these prosencephalic ganglia has a surface perfectly free, that is, devoid of medullated attachments—and other aspects, which present the termini of centric and peripheric strands. Thus, the ventricular aspect of the caudate, together with the base of the lenticular wedge (insular aspect), are alike smooth and devoid of medullated connections; whilst the opposed surfaces, separated by the intervening capsular fibres, as well as the basal or inferior aspect of the lenticular nucleus, are the surfaces for the termination and departure of the numerous medullated connections of this with distant regions. Since the *lenticular* body lies *beneath* the internal capsule, its temporal extremity is separated for some distance by that formation from the temporal extremity or *cauda* of the intraventricular nucleus. Posteriorly, however, they approach each other, and bridges of grey matter connect them, separated by medulla. They are also separated here by the centric extension (brachium) from the external geniculate body; and, finally, along the roof of the descending cornu these two temporal extremities fuse together, forming the lower segment of the *surcingle.*

The constitution of the surcingle, therefore, is different in its upper and lower arc, being purely an extension of the *innermost* nucleus above; but formed out of the fused temporal extremities of both prosencephalic nuclei below. It cannot fail to impress the student that the ganglionic structures and their extensions, so far described, encircle in a series of loop-like formations the medullated core which passes from the spinal cord and medulla upwards as peduncles and capsule to the cerebral hemispheres.

First, there is the mesencephalon, the quadrigeminal bodies, each throwing downward its loop-like *fillet* or *lemniscus;* and throwing upwards its centric arm in the form of the *brachia.* At a higher level, the thalamencephalon shows us the optic thalamus astride the posterior edge of the internal capsule, arching backwards around it to form the roof of the descending horn of the lateral ventricle; whilst its centric extensions pass upwards to the cortex from its outer surface beneath the tail of the caudate. Then still higher we get the arc of the caudate body astride the anterior edge of the internal capsule with its long tail-like loop also passing down the roof of the descending cornu in conjunction with that of the lenticular: whilst still further outwards is the mass of the internal capsule becoming free as coronal radiations to the various parts of the hemisphere. For descriptive purposes it is convenient to distinguish between **upper** or **ventricular,** and **lower, cornual** or **temporal arc** of the *surcingle:* the **caudal** extremity, the **body** of the caudate nucleus, and its **caput** directed towards the base: whilst we also speak of its **ventricular** and **capsular** aspects.

In like manner the lenticular nucleus has its frontal, its temporal, and peduncular or crustal extremity; its insular aspect (or base of wedge), its inferior aspect, its capsular aspect. So also the capsular constituents may also bear the convenient terminology—**cortico-striate** and **cortico-lenticular** fasciculi for the centric bundles: **pedunculo-striate** and **pedunculo-ventricular** for the peripheric bundles: **direct peduncular** for those uninterrupted by the prosencephalic ganglia.

Stria Terminalis.—A glistening white band of fibres, strongly contrasting with the adjacent grey cauda, varying from one to two millimetres in diameter, lies along the inner border of the tail of the caudate body throughout its whole length, extending from the tip of the temporo-sphenoidal lobe along the roof of the descending cornu, and along the upper arc of the surcingle betwixt it and the thalamus, as far as the anterior end of the latter. Inferiorly it is distributed to the **amygdaloid nucleus**; and by Meynert is regarded as arising from the head of the caudate nucleus. Schwalbe, on the other hand, regards this conclusion as dubious, and expresses the opinion that it possibly has no connection whatever with the ganglion. By other authorities—Meckel, Arnold, Jung, and Luys—this arciform band has been presumed to terminate in the descending pillar of the *fornix*. This lengthened arciform structure, which has been also called the **tænia semicircularis,** would appear from its greater proportionate development to be an important structure in the brain of rodents; in the dog, on the other hand, it is comparatively insignificant in size. In the rabbit in can be clearly seen to consist of a superficial and deeper fasciculus at its termination, and to be connected throughout its course with the caudate nucleus: the latter arches downwards behind the anterior commissure just to the inside of the lower margin of the internal capsule, as seen in vertical sections: these fibres appear to terminate in the area at the base known as Gratiolet's **olfactory area**—embraced, in fact, within the **trigonum olfactorium.** It would be hazardous to affirm that none of the superficial fibres enter the descending pillar of the fornix, as stated by several authorities; but in the rodent it appears easily demonstrable that this fasciculus, in great part at least, enters the anterior commissure from behind in such a direction as to ensure *decussation*, and so bring the hippocampal region and caudate nucleus into crossed relationship with the olfactory bulb.* We shall refer to this connection further on when dealing with the relationships of the anterior commissure.

Lenticular Nucleus.—Although in section both vertical and horizontal, this ganglion exhibits a distinctly wedge-shaped contour, its name of *lenticular* is justified upon inspection of its outer or insular aspect. This can only be done by freeing it of its medullated connections—an operation readily effected either by dissection or the brush, the ganglia being held beneath water, whilst the medullated investments are dissected off or brushed away. In this manner it is easy to isolate the two ganglia of the prosencephalon attached to each other, for the purpose of recognising their fundamental configuration.

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* See "Comparative Structure of the Brain in Rodents," by the Author, in the *Philosophical Transactions*, part ii., 1882, p. 730.

The lenticular, then, appears to be a distinctly lens-shaped body, especially if looked at from above, where a section of the internal capsule at the foot of the **corona radiata** separates it from the caudate nucleus within. It will then also be apparent that the caudate, applied at first to the inner side of the internal capsule and lenticular, mounts higher and higher so as to lie with its attenuated tail *upon* the former and *above* the latter. The smooth lens-shaped exterior of this structure is overlaid by a medullated investment—the external capsule, loosely applied to it, occasionally the site of hæmorrhage, which breaking into the intervening tract, separates it from the surface of the lenticular.

External to this capsule comes the **claustrum,** and, lastly, the medulla and cortex of the **insula** or **island of Reil.** Upon section this ganglionic body shows a well-defined triple segmentation—distinguished by the grey aspect of the outer, and the increasing pallor and tawny pigmented aspect of the more internal segments; the innermost and largest which is notably pale and pigmented being designated the **globus pallidus** (fig. 8). These three divisions are not merely distinguished by their difference in colour, but are separated by well-marked dissepiments called **laminæ medullares,** which, as thin medullated partitions, descend from the internal capsule down to the basal aspect of the brain, lying concentrically to the insular aspect of the ganglion: two and sometimes three such dissepiments exist. The medullated fibres forming these dissepiments, and arising from the internal capsule, bend inwards at different points to form radial fibres, all directed towards the peduncular end of the wedge; a certain proportion, however, completely traverse the lenticular as laminæ medullares; and, escaping at the base of the ganglion, pursue their course towards the crusta, as a sort of capsular sling, covering the base of the lenticular and forming one layer of the so-called *ansa lenticularis* already referred to. Since each segment is traversed not only by fibres originating in the cells of its territory, but also receives those passing into it from an outer segment and its medullary lamina, it follows that the narrow or peduncular end of the wedge becomes constituted by a closely packed system of medullated fibres where they enter the crus cerebri; and it is this preponderance of fibre over grey matter which gives to the inner segment (globus pallidus) its characteristic pallor.

THE ENCEPHALON AS A WHOLE.

Comparative and Embryological.—The earliest indication of a brain in the vertebrate series consists in a slight bulb-like dilatation at the end of the neural tube. This is all that ever occurs in the lowest form of vertebrate animals—the **amphioxus** or **lancelet,** which therefore presents as a permanent structure the earliest, but transitory, phase of development, through which all higher vertebrata pass, even to man himself. A step higher, the **lamprey** exhibits a large pyriform dilatation of the neural tube, and retains for a long period this rudimentary form, which, however, in comparison with its spinal system, bears to it scarcely a higher proportionate size than do the cephalic ganglia of insects to their ventral ganglia.

Still higher in the vertebrate series, in **fishes** and **amphibia,** we find that this bulb-like distension of the neural axis becomes very early transformed by transverse constrictions of the former elongated bulb, into a series of five pairs of vesicles, which lie in linear series, one behind the other, and which are reproduced in every form of vertebrate, higher in the series, at a certain stage of its developmental history. These five vesicles represent what in higher animals become respectively the **fore-brain** or **cerebrum,** the **twixt-brain,** the **mid-brain,** the **hind-brain,** and the **after-brain.**

These several parts in fishes and amphibia represent elements of the higher vertebrate brain, which remain permanent in them, but subject to most diverse modifications in structural complexity and in relative preponderance of one or other segment. The fully developed brain in fishes presents great variety in the relative size of the individual lobes. In the first place, the early differentiation between the vesicle of the twixt-brain and mid-brain becomes obscured in most fishes, so that the fully formed organ shows us but four gangliated swellings, lying one behind the other in series, and representing (1) the **cerebral hemispheres;** (2) the **optic lobes;** (3) **the cerebellum;** (4) the **medulla.**

The two former, as seen in the brain of the perch, are disposed in pairs, whilst in front of the cerebral hemispheres we see yet two small bulbous swellings, from which arise the olfactory nerves, and which are called, therefore, the **olfactory ganglia.** These ganglia are absent in the Shark, Skate, Whiting, &c., and are replaced by an elongated peduncle capped at the extremity by the ganglia as in man.

The cerebral hemispheres in **fishes** are usually smaller than the optic lobes—*e.g.*, in the Whiting, Carp, Pike, Perch; but in the Shark, the Skate, the Lepidosiren, and others, they very greatly exceed these lobes in their dimensions. Behind the cerebral hemispheres appear the optic lobes, which in the fish, it must be remembered, represent the thalamencephalon (thalamus and third ventricle) as well as the mesencephalon (or in man what corresponds to the corpora quadrigemina). Thus, if we turn to the brain of the perch, we see in front the two small olfactory lobes, followed by a large pair of cerebral hemispheres, and these in their turn by the still larger pair of optic ganglia, with a small tubercle projecting in front between them and the cerebrum. This latter body is the **pineal gland,** indicating the neighbourhood of the thalamencephalon, with which it is connected. At the base the same structures are seen in front; but the optic lobes present two peculiar lobulated bodies called **hypoaria** or the **lobi inferiores,** whose significance is unknown: they are peculiar to fishes. From the centre projects the pituitary body, whilst the optic nerves are seen to originate from the base of this ganglion, and cross (without decussation of fibres) to the opposite sides. In **insects** these optic lobes represent the chief part of their cephalic ganglia.

Behind the optic lobes comes a single tongue-like lobe—the cerebellum—the size of which apparently bears a direct relation to the power and muscular activity of the fish. Thus the rapacious shark has an enormous cerebellum, whilst in the more sluggish fish, it is relatively small; in osseous fishes it is usually considerably below the size of the optic lobes. The last division of the brain is the medulla, lying immediately behind and beneath the cerebellum, mapped off from the spinal cord by its somewhat larger size, and the origin of numerous important nerves.

In **amphibia,** the brain presents a smaller cerebellum than in fishes, corresponding with their more torpid habit.

The **reptilian** brain differs from that of fishes, chiefly in the smaller relative size of the optic and olfactory lobes and cerebellum—the latter being often a mere delicate transverse band across the upper part of the medulla; and in the relatively large size of the cerebral hemispheres, which partly overlap the optic lobes and exhibit a distinct striate body. The cerebellum is especially large in the crocodile. The hemispheres are connected as in fishes by an anterior commissure.

In **birds,** the cerebral hemispheres exhibit a great developmental advance. They are very large, and cover more or less completely the optic lobes. The cerebrum contains a distinct cavity, corresponding to the **lateral ventricles,** and communicating with the **hollow peduncle** of a small **olfactory lobe** in front. From the floor is developed a gangliated mass—the **corpus striatum.** The optic lobes (corpora bigemina) are two smooth, rounded, egg-like bodies, just apparent from beneath and behind the hemispheres; widely separated, but communicating through a hollow passage which also leads into a channel between the third and fourth ventricles. From below we see the optic nerves arise and distinctly decussate across the middle line.

The cerebellum is also of large size, but chiefly consists of the middle lobe.

The germinal area of the mammalian ovum reveals at an early stage the **medullary groove,** as a longitudinal and gradually deepening channel in the fore-part of this area—at first of uniform diameter throughout, but soon becoming widest at one end—the cephalic. The groove itself results from the thickening of the outer germ layer or **epiblast** in two parallel linear streaks, corresponding in direction to the long axis of the embryo. The thickening of these parallel ridges proceeds until the groove thus produced is covered in by the bending across and coalescence of these its walls—the so-called **medullary folds.** Thus, the medullary groove becomes converted into the closed canal destined to become the cerebro-spinal cavity, and now termed the **neural canal.** At its fore end, this canal is dilated into a bulb or vesicle—the **primary cerebral vesicle**; whilst shortly afterwards two other vesicles, separated by constrictions of the neural canal, form along this end of the canal immediately behind the first vesicle. These three vesicles, placed one behind the other, lie in a straight line with the axis of the neural canal, and are termed respectively the **vesicles** of the **fore-brain,** the **mid-brain,** and the **hind-brain.**

Vesicles of the Fore-brain.—From the first of these, a lateral bulging on either side becomes soon apparent, and, steadily increasing, is at last merely connected with the former by a narrow constricting neck or tubular stalk. These give origin to the more important structures of the eye and are termed the **optic vesicles.** By an exactly similar process, two other lateral bulgings from the fore-part of the first cerebral vesicle become differentiated therefrom; and these are destined by rapid growth and development to become the most important and conspicuous parts of the cranial contents. They form the **cerebral hemispheres** or **prosencephalon.** At the second month of intra-uterine life, they are mere insignificant ampullæ, of somewhat oblong form; but even now presenting a short tubular extension from their tip, which is the rudiment of the **olfactory lobe.** The remaining portion of the primary vesicle in its median position enters into the constitution of the parts around the third ventricle. It has consequently been named the vesicle of the third ventricle, or the **tween-brain,** or from giving origin to the optic thalami—the **thalamencephalon.** Hence the two pairs of vesicles, the optic and hemispheric, have their genetic origin from the thalamencephalon, and a direct connection between these structures is maintained during all later stages of development. The cerebral hemispheres, as offshoots from the primary cerebral vesicle, are hollow vesicles, communicating with each other and with the cavity of their parent vesicle, the third ventricle, by means of the **foramen of Monro.** As the walls of the hemispheric vesicle gain in thickness, its cavity becomes of course more and more encroached upon; yet, for a long period during uterine life, the growth of this vesicle is so rapid that its cavity is of great size; this cavity forms therefore a relatively capacious ventricle—the *lateral ventricle.* In the outer and lower wall of the hemispheres thickening proceeds to the extent of forming a large ganglionic mass, the *corpora striata* or **ganglia** of the **fore-brain.** These ganglia, it is to be noted, are not *directly* derived from the primal neural tube, but from an offshoot of the latter—the hemispheric vesicle. The posterior moiety of the first cerebral vesicle in like manner exhibits a thickening of its walls, which form the ganglionic mass of the *optic thalami*, connected behind by the *posterior commissure*, just above which a small median projection forms—the *pineal gland:* its floor upon the other hand, sends downwards a conical projection—the *infundibulum*, which, later on, unites with the **hypophysis cerebri** or **pituitary body,** immediately over the pharynx or extreme end of the alimentary canal. The funnel-shaped extension of the third ventricle is by some regarded as the representative of the **neuro-enteric canal,** which establishes connection directly between the cerebral and caudal extremities of the alimentary canal and the central canal of the cerebro-spinal system. The upper part or roof of the thalamencephalon becomes thinned out into a mere lamella of pia mater, covering the third ventricle as the *velum interpositum.*

The **second cerebral vesicle** or **mid-brain** exhibits no such budding off of secondary parts as does the primary vesicle; its upper hemisphere becoming thickened, ultimately forms the *quadrigeminal bodies:* its lower hemisphere or floor in like manner develops into the *crura cerebri* or *cerebral peduncles:* whilst the central cavity thus encroached upon becomes eventually reduced to an exceedingly narrow channel, continuous in front with the third ventricle, and behind with the hollow of the third cerebral vesicle—this channel is the *sylvian aqueduct* or *iter a tertio ad quartum ventriculum.* The ganglia of the mesencephalon or corpora quadrigemina are not completely differentiated until the sixth or seventh month of intra-uterine life. At the sixth month, a *vertical* groove severs the vesicle into lateral pairs; at the seventh month, a *transverse* groove separates the upper pair or *nates* from the lower pair or *testes.*

The **third cerebral vesicle** or **hind-brain** becomes differentiated into two segments—an upper, immediately behind the corpora quadrigemina, from which is derived the cerebellum, pons, and upper part of the fourth ventricle; and a lower, forming the lower half of the fourth ventricle and medulla oblongata. The roof of this lower segment thins away to such a degree that, like the velum interpositum, it also becomes a mere membrane closing in the ventricle at this site. The upper segment is termed from the cerebellum the **hind-brain** or **epencephalon,** the lower segment the **after-brain** or **metencephalon.**

The Cranial Flexures.—At a very early date, the first cerebral vesicle begins to curve downwards around the extreme end of the **notochord,** until, from being in a line with the latter and longitudinal axis of the embryo, it becomes placed vertically at right angles to this axis. An angle or bend thus occurs between it and the middle vesicle, which, in its turn, becomes most prominent and in a line with the notochord. A second bend in consequence of this flexure also occurs between the middle and the posterior vesicle, or that portion of it which becomes the **cerebellum**; a third takes place between the latter and the hinder half of this vesicle, which becomes the **medulla oblongata**; and yet another between this region and the commencement of the Spinal Cord. These cranial flexures, which occur between the first cerebral vesicle and its derivatives, the Quadrigeminal bodies, the Cerebellum, the Medulla oblongata, and the Cord, are stated by Tiedemann to take place about the seventh week.

In the further process of development the **cerebral hemispheres** or **prosencephalon** enlarge wholly out of all proportion to the hinder parts of the neural tube, so that the quadrigeminal bodies which hitherto, as in animals, have had a relatively large bulk compared with the cerebrum, become now placed quite in the shade beside the rapid advance made by the cerebral hemispheres. They extend upwards and backwards, covering and concealing the thalamus by the *third* month, the corpora quadrigemina by the *sixth* month, and the cerebellum by the *seventh* month of intra-uterine life. Long prior to these last changes—in fact, about the *fourth month*, a slight depression appearing on the outer aspect of each hemisphere midway between its anterior and posterior extremity, indicates the position of the *sylvian fossa*; and were a horizontal section of the hemisphere carried through this depression we should find the walls of the vesicle within much thickened at this point, the thickened mass protruding into the central cavity as the rudimentary **striate ganglia.** This fossa, which is seen early in all mammalian brains, becomes the **insula, island of Reil,** or **central lobe,** whilst the cortical structure, thickening around it, forms a distinct fissure, the sylvian fissure, whose upper and lower margins encroach upon and cover the "island" from view. Up to the fifth month, however, the fissure of Sylvius remains patent, exposing the island to view. The fissures of Rolando often appear about the end of the fifth month, whilst together with the fissures of the frontal lobe, they are prominent objects on the surface of the hemisphere at the termination of the sixth month of uterine existence. About the same time also appears the internal parieto-occipital fissure on the inner aspect of the hemisphere, mapping off the occipital from the parietal lobe on its median aspect. This fissure in its descent meets the hippocampal fissure at the point where its posterior extension forms the so-called calcarine fissure.

THE CEREBRAL CORTEX.

The fundamental divisions of the encephalon, or brain, in mammals are identical with those existing in the whole vertebrate series of skulled animals (*Craniota*). The early history of embryonal existence is alike for all—each animal higher in the scale, even the highest—man, reproducing at an early period of embryonic development, as a transient condition, the features permanently stamped in those of a lower grade. The infinite degree of complexity ultimately obtained by the mammal's brain is prefigured by the forms assumed in the lower classes of vertebrata, and depends, for the most part, upon the preponderance of certain divisions of the encephalic mass over others, and also upon the growing complexity of individual parts, either as the result of increasing differentiation of existing structures or the addition of supplemental parts in the form of gangliated masses or fibrous tracts. In the mammal's brain we find the first condition exemplified in the complicated convolutionary surfaces of the cerebrum; in its division into lobes and lobules; in the wondrous complex structure of its cortex. We find the second exemplified by certain ganglionic outgrowths from the original brain-vesicles, and along its fibrous tracts in the large striate and thalamic ganglia, in the lenticular body, the optic basal ganglion, the corpora geniculata, and others; and yet again in the extreme development of the callosal and other connecting systems of the brain-mass. The endless diversity of richly-convoluted brains in mammals introduces no feeling but that of confusion to the mind of one who has not studied the cerebrum in its various forms as presented by the whole range of mammalian animals; in fact, a comparative investigation can alone teach the student the significance of its complicated mantle, and help him towards recognising homologous parts in the series.

Great advance has been made towards this end by the labours of Gratiolet, Ecker, Turner, Broca, Huxley, who, amongst other valuable results, have introduced a definite nomenclature which reduces to precise terms, universally intelligible, the statements of different workers in this department. Another field of enquiry has added rich results in the same direction—the physiological. From that epoch in the historical development of Nervous Physiology when it was discovered that the cerebral cortex was excitable to electric stimuli, with patient toil have questions been put and answers received by this method of research, and a mapping out of the complicated fields of the cortex into physiological territories established by Ferrier, Hitzig, Horsley, and others. Another field of enquiry has received but scant attention, yet it is one which an accurate scientific knowledge of the cortex must make its own: I refer to the histological structure of the whole cerebral mantle in its various districts, as supplemental to the coarser examination of its medullated tracts by the cleavage methods of Gratiolet, &c. It is but a natural *a priori* conclusion that differentiation in cerebral *function* implies likewise a *structural* differentiation, and that this latter is one of qualitative as well as quantitative value. We naturally look for an alteration of structure as well as disposition of individual elements, and the increasing heterogeneity of such individual parts we regard as the logical outcome of the law of evolution.

Thus it is we expect the physiological areas ascertained by Ferrier to exist in the brain of the monkey and other animals to exhibit a structural differentiation characteristic of those parts, and hence to be helpful in the recognition of analogous regions in other orders. If it can be established that areas, whose functional endowments are familiar to us, present *uniformly specialised anatomical* features, we may reasonably conclude that other structurally differentiated areas, whose functions are unknown to us at present, have each and all of them diverse endowments. An attempt to delineate the homologous areas of the cortex in different orders of mammalia by simple inspection would (on *a priori* grounds) only lead to failure; indeed, errors have already been frequently committed with respect thereto: the method of physiological experimentation can alone lead to conclusive results. But, meanwhile, we should not neglect the important consideration of making ourselves acquainted with the intimate structure of the cortex, which also has its own special significance, and which would frequently enable us to avoid the error of drawing our analogies from a mere superficial resemblance of the convolutionary surface.

The grey matter enveloping the exterior of the cerebral hemispheres, the **cortex cerebri,** merits our most careful study, as being pre-eminently the site of those deranged functions and pathological processes which express themselves in mental disease. Whatever the limits our definitions compel us to impose upon the sphere of consciousness, all are agreed that here, in the wondrous web of nerve cell and nerve fibre, take place those activities which underlie the conscious states we denominate **mind.** It becomes, therefore, an essential part of the training of the student of Mental Disease to render himself practically acquainted with the structure and functions of the cerebral cortex—the "tissue of mind." This grey envelope which receives the terminal extensions of the ingoing channels of communication with the outside world, on the one hand, and forms, on the other hand, the origin for the outgoing currents of the same, plays a supreme *rôle* in the nervous hierarchy, and to it all other centres of grey matter are subordinate. In the human brain, the cortex is continuous all over the hemispheres, dipping into the various sulci between its convolutions, and terminating at the median constricting ring through which the brain-stem of the peduncles and the great commissural tract of the corpus callosum pass.

The distribution of the surface into intricate convolutionary folds, such as occur in man, is at the outset somewhat perplexing to the student. Far better is it in his case to study the brain of some of the lower animals, which present a smooth non-convoluted surface, and gradually extend his enquiries to the convoluted brain of higher animals, and, lastly, of man. Beginning thus with the simpler forms of life, he is better able to appreciate in the wonderful architecture of even these simple brains the profound intricacy of the nervous centres of man: he meets with fewer obstacles at the outset to discourage his attempts, and he lays the foundation for a comparative knowledge of the brain, which will be of inestimable value to him in his subsequent studies.

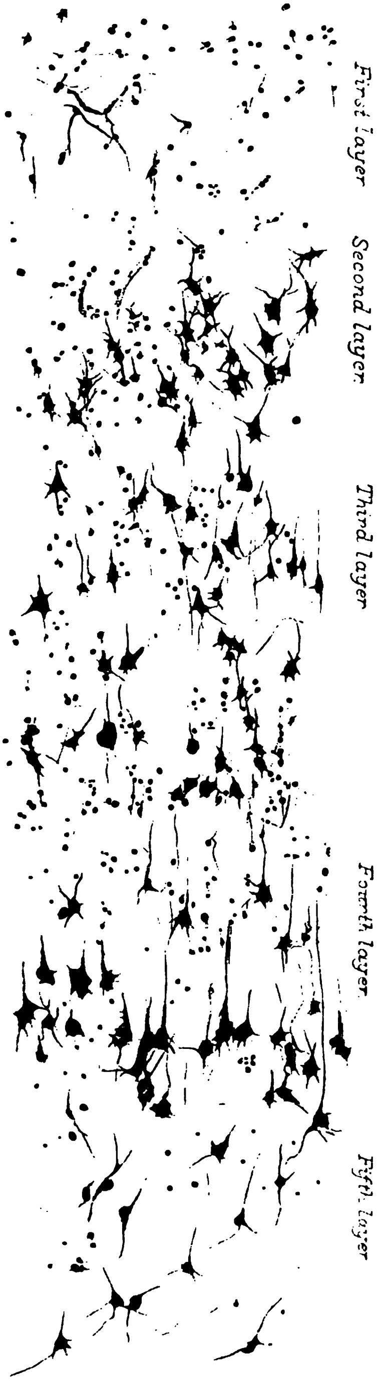
We shall adopt this plan in the following study of the cortex cerebri, and commencing with the brain of a small Rodent, which is a smooth-brained animal, take the Rat as our illustration.

Histological Elements.—In the first place, what are the elementary constituents of the cortex? This is a necessary question to dispose of, ere we pass to their local distribution, regional preponderance of certain elements to the exclusion of others, and their relationships to underlying tracts of medulla. The elementary constituents of the cortex are

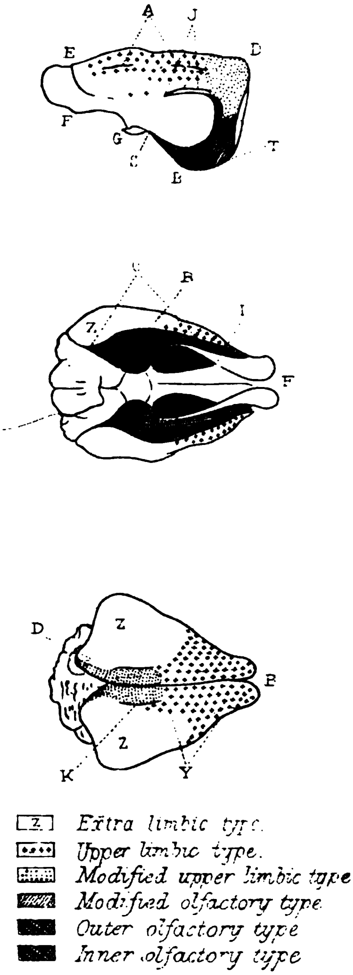
(*a*) Nerve cells.
(*b*) Medullated and non-medullated nerve fibres.
(*c*) Connective meshwork of "neuroglia cell and fibre."
(*d*) Blood-vascular supply.
(*e*) Lymphatic supply.

(*a*) Nerve Cells.

Minute Structure.—The nerve cell, in contradistinction to the nerve unit, or neuron, is an irregular mass of protoplasm, in a large proportion of instances approaching a more or less spheroidal contour, but frequently elongated, fusiform, triradiate or multipolar. Its contour is dependent upon the site and number of its branches, and thus a bipolar cell will assume a spindle form, whilst a multipolar cell will partake of an amœboid contour. Whatever the form of cell, however, certain constant characteristics are displayed in all alike in common with the cells of all animal tissues. The nerve cell *invariably*



"Motor Type"
in extra-limbic area of Rabbits Brain
a frontal pole of hemisphere x 200.



Brain of Rabbit
Mesial aspect, basal aspect & Vertex
Distribution of various laminated types of Cortex.

Bale & Danielsson, Ltd. Sculp.

contains a nucleus—usually of large proportionate size compared with the cell itself, and of a structure essentially differing from the cell protoplasm surrounding it. Apart from *morphological* differences a *chemical* distinction exists betwixt nucleus and the cell body, which is of critical importance in the physiological phases through which the cell passes, and to which, later on, we shall refer. The nerve cell has no definite wall or membrane, such as we find in the vegetable cell, although in pathological states such *formed* material is often to be recognised; on the other hand, the nucleus is possessed of a distinct membrane which sharply demarcates it from the surrounding cell protoplasm, and which, in certain cells, may assume a considerable density. Usually placed centrally within the cell, the nucleus may, in certain morbid conditions, be transferred to the side or be dislocated to the apical pole of the cell, as is so often seen in the pyramidal cells of the cortex.

The Cytoplasm.—The protoplasmic contents of the cell apart from the nucleus is spoken of as the cytoplasm; whilst that of the nucleus is known as the karyoplasm. The cytoplasm is by no means clear, homogeneous and structureless, as direct observation of the fresh unstained cell would lead us to infer; but is, like the corresponding karyoplasm, of highly complex structure. A very delicate fibrillar structure pervades the whole cell, becoming continuous with close set parallel fibres, which pass into the several processes given off from the cell body, and to which they give a finely striated appearance. As these fine striæ pass into the cytoplasm from the cell processes they diverge and form, according to most authorities, a sort of reticulum, or delicate meshwork or sponge (*spongioplasm*) which supports peculiar granules or rod-like bodies known as the chromophil granules of Nissl.

The achromatic fibrillar structure or cytoreticulum has been recognised by Benda, Dogiel, Nissl, and others, in nerve cells, and has been identified by them with the filar mass of Fleming. Although the achromatic fibrils are traced into all the processes of the cell, the granules of Nissl (or "chromatophilic" substance) do not enter the axon, as was shown by Benda in 1886, whilst they extend freely into the large dendrons. The "chromophilic" masses of Nissl, usually rod-like, tetrahedral, polyhedral or granular in aspect, may be disposed concentrically around the nucleus, or parallel to the cell contour: whilst, converging towards the dendrons, they course through the latter in parallel streaks. They show a very strong affinity for certain dyes, especially the colour derivatives of di-phenylamine—such as methylene blue and thionin, and so also for toluidin blue. With the former of these stains, as by Nissl's method, the granule masses are deeply coloured, as are also the nucleoli, whilst the nucleus remains unaffected. Nissl has classified all nerve cells according to whether the granule masses are arranged :—

(1) In a reticulated pattern or meshwork (Archyochromic);
(2) In elongated spindles, streaks or knots (Stychochromic);
(3) Or without any apparent order or groupings (Gryochromic).

These granules are regarded as incrusting the cytoreticulum, filling up more or less its meshwork. When they are approximated by the diminution or shrinking of the cell they show deeper staining, and the cell is regarded as in a **resting** stage (pyknomorphic); when more widely diffused and the cell increased in size, it is regarded as evidence of functional activity (apyknomorphic). Since the term "resting," however, has long been applied to the stage preceding mitosis, it should be discarded here; as also should the term "chromophilic" as applied to the granules of Nissl, since it is likely to be confounded with the chromatin masses of the nucleus, with which it must *not* be identified.

Lugaro * regards Nissl's granules as identical with Fleming's "interfilar" mass, and, so far as cell function is concerned, as having a passive *rôle.* †

The filar structure is supported by the more fluid, transparent **ground-substance** or **hyaloplasm.** Reasoning from analogy we should presume that this reticulated structure is itself composed, as in other animal cells, of extremely minute granules—the so-called "microsomes"; and that they are probably directly continuous with the similar structures in the nucleus. If this be so, the nucleus evidently does not lie free within the cell.

The cytoplasm of nerve cells is very frequently the site of pigment granules, which, in the pyramidal cells, in physiological states invariably assume a position at the base of the cell; but in pathological states may be very widely diffused throughout the cytoplasm.

Granular proteid matter is also found here in disease, as well as fatty granules—products of cell metabolism. These, together with vacuoles, form the principal lifeless structures in the cytoplasm.

The nerve cell is, according to Lenhossek, not an exception to other tissue cells as regards another all-important constituent of the cytoplasm: this is the so-called "centrosome"—a single or double granule exceedingly minute, scarcely larger than a microsome, deeply stained by certain reagents, and distinguished by its forming the centre of a peculiar area of the cytoreticulum called the "attraction-sphere." Usually in close contiguity to the nucleus, it forms the starting-point for all those mysterious changes which result in division of the cell and reproduction.

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* *Rivista di Pathol. nerv. e. Ment.*, vol. i., Jan., 1896.

† "Sulle Modificazion delle Cellule nervose nei diversi Stati functionali." *Lo Sperimentale*, vol. xlix., Aug., 1895.

Ayers has identified the centrosome in the nerve cell of the electric lobe of the Torpedo, and has noted the frequent presence of two, or even three, nuclei. He also clearly traces the division of such nerve cells in the Torpedo brain—although the usual teaching has been that after the third month of fœtal life the neuroblasts no longer increase in number but in size only (*His*), and that the appearance of axons and dendrons is final for all further cell division.*

Nucleus.—As before stated, the nucleus is a constant feature of all nerve cells; usually spherical or oval in contour, sharp edged from its possession of a limiting membrane, it contains within it a **ground-substance** or karyolymph pervaded by an extremely fine thread-like reticulum—the linin reticulum, with minute granules along its course, and continuous with the nucleus membrane and the cytoreticulum of the cell. But, beyond this, a much coarser structure readily stained by certain dyes, and which varies much both in amount and in appearance at different periods of the cell life, is a still more prominent feature. This latter material may form an irregular meshwork or accumulate in large granule masses, and is peculiarly characterised by staining of an intense depth with *basic* aniline dyes. From the latter feature it is called "Chromatin," and must be carefully distinguished from the "chromophilic" granules of the cytoplasm. Within the nucleus is usually found a spherical body stained deeply by plasma dyes, and, so, reacting like the cytoplasm: this is the so-called "nucleolus."

Dendrites and Thorns.—The dendrites of the cortical nerve cells are beset in most cases, if not in all, by delicate thorn-like projections, each tipped with a minute bulb, which, together with the stalk, is stained deep black by the silver-chrome and mercury methods of Golgi and Cox. They resemble the gland-bearing hairs of the vegetable kingdom rather than true spines, thorns (*épines*), and commence where the first branchings of the main dendron occur. They give the dendrites a rough, hispid appearance, which at once serves to distinguish the protoplasmic processes from the smooth contoured axon, the latter being always devoid of such projections. These thorns or gemmules, as they have also been termed, vary considerably in length at different sites along the dendrite—Hill states from 1 μ to 8 or 10 μ; the "longest being found in the granules of the olfactory bulb." †

The function assigned to these thorns has been that of affording multiple points of contact for the nerve fibril, naked axons, and terminal arborisations, which pass athwart the dendrites. On the other hand, the theory held by Hill is expressed by him in these words:—"The thorn is really the cell end of an unstainable nerve filament surrounded by a film of staining cell plasm."* If this position be confirmed, the whole doctrine of conduction of nervous currents by *contact* falls to the ground and the *continuity* doctrine of Gerlach would, with certain modifications, be re-established.

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* Morphol. Labor. Univ. Missouri, Columbia, March, 1896.

† "Note on Thorns," *Brain*, vol. xx., p. 133.

Chemical Constitution.—The linin network of the nucleus and the cytoreticulum are identical in constitution, and, as Wilson indicates, this identity is well established by the fact that the spindles of the mitotic figure in certain rare cases may arise *within* the nucleus, as where we have an intra-nuclear centrosome. This achromatic network has, of course, a chemical constitution entirely differing from the chromatic network or chromatin masses, as indicated by staining reactions. The minute granules along this network stain readily with *acid aniline* dyes, such as acid fuchsin, eosin, rubin, and Congo red; granules so reacting are termed Oxychromatin granules. On the other hand, the granules and masses of chromatin are termed Basichromatin since they stain vigorously with *basic aniline* dyes—viz., methyl green, saffranin, Bismarck brown. The Basichromatin or Chromatin of biologists is now almost definitely ascertained to be identical with the **Nucleins** (*Miescher*) complex organic compounds of nucleic acid and albumen, in which the proportion of nucleic acid may vary very greatly.

It has been ascertained by direct experiment that mixtures of nucleic acid and albumen when treated by methyl green and acid fuchsin take up a green, bluish-violet, or red coloration *according to the amount of Nucleic acid present*—the basic dye always predominating with the larger amount of nucleic acid (*Malfatti*). The deeper staining of the basichromatin, therefore, indicates the presence of larger proportions of nucleic acid; and this seems to be indicated in certain pathological states of the cell nucleus as in hydrophobia, also in certain physiological phases of the cell life—as in the case with the chromosomes just prior to and during mitosis, and again with the nucleus of the spermatozoon, which, judging from the intensity of the reaction, must be nearly pure nucleic acid.

Nucleic acid is remarkable for the large amount of phosphorus combined with it—pure nucleic acid having over 9 per cent. of phosphorus and no sulphur; whilst the nucleins found in cell nuclei vary in their amount of phosphorus from 3·2 to 9·6 per cent. In fact, we have in the percentage of phosphorus the means for estimating the purity of nucleic acid in basichromatin. The chromatin network embraces also many granules identical with the oxychromatin granules of the **achromatic** network; and, since every phase of coloration from basic to acid reaction occurs in these granules when treated with differential dyes, it is believed that the chromatin is directly convertible into oxychromatin by the loss of its greater proportion of phosphorus—in other words, a lower member of the nuclein series results (*Wilson*).* All late researches seem to point to the fact that in the growth of the chromatin masses, and in the loss of their staining capacity for basic dyes, we have evidence of the taking up of albuminous material and the giving up of phosphorus, and that this occurs during periods of great constructive activity of the cytoplasm : whilst, with the diminution in size of the chromosomes and their more intense staining, such synthetic metabolism is at its lowest ebb.

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* "The Chrome-Silver Method," *Brain*, 1896; also *loc. cit.* vol., xx.

It is of great interest to note that the nucleins which play so important a part in the economy of the nerve cell (as well, indeed, as in all cells of both vegetable and animal tissues) have as their decomposition products the highly nitrogenised compounds called the xanthin bodies—viz., adenin, guanin, xanthin, and hypoxanthin, and which, as illustrated by the following table, are the direct precursors of uric acid which probably represents the total result of nuclein decomposition :—

Adenin, ...	$C_5H_5N_5$
Guanin, ...	$C_5H_5N_5O$
Hypoxanthin, ...	$C_5H_4N_4O$
Xanthin, ...	$C_5H_4N_4O_2$
Uric acid, ...	$C_5H_4N_4O_3$

Physiological and Pathological.—As yet, we are far from being able to give a decisive opinion upon the physiological significance of the cytoreticulum and the chromatophil granules of Nissl. Authorities are much at variance on this point, and the results of experimental research are frequently of a conflicting nature. It may not be premature, however, to give here the more important and seemingly conclusive results obtained. In the first place, the granules of Nissl, or so-called "Tigroids," may be regarded as representing Fleming's **inter-filar mass:** the unstained, achromatic substance corresponding to his **filar mass.** In the next place, these granules form no part of the *conducting* mechanism of the neuron—they play a more passive *rôle.* Whether they be nutritive material, as Lugaro suggests, or whether (as we opine) they in some manner intensify the nervous discharge along the conducting strands, they take no *direct* part in the conveyance of the nerve impulse, which almost certainly is the attribute of the achromatic or filar network (cytoreticulum). Although, as it were, superadded to the true anatomical conducting mechanism of the neuron, they by no means act like the lifeless bodies which are so often found in the cell protoplasm generally; on the contrary, they are peculiarly sensitive in their reaction to various

* "The Cell in Development and Inheritance," *Columbia Univ. Biol. Series,* iv.

agencies, although Lugaro would maintain that they bear a direct relationship to the size of the cell rather than that they vary with its functional conditions. A great mass of evidence is, however, now forthcoming to show that these granules, both during certain physiological phases of cell life and as the result of experimental irritation, traumatism, or pathological change, undergo very notable and extensive alteration.

Chromatolysis.—The most familiar change observed consists in a peculiar *diffusion* of the chromophil granules, which, instead of lying closely around the neighbourhood of the nucleus as in pyknomorphic cells, are scattered peripherally, the granules being more widely severed, whilst the nucleus is often displaced towards the periphery of the cell. Occasionally, a clear zone surrounds the nucleus still central in position; or, again, the granules may be *generally* diffused and broken down into a fine pulverulent condition, giving the cytoplasm a punctuated aspect. In other instances the granules may not be so much altered in form as displaced outwards, and surrounding an unstained vitreous, homogeneous looking substance.* Chromatolysis may be a purely functional change—the granules may again resume their former size, position, and staining reaction; on the other hand, it may be the precursor of complete loss of normal structure and break down of the neuron.

Fatigue.—Attempts have been made by electrical excitation of the nervous system of the frog, the dog, and cat (*Hodges*) to ascertain the effect of fatigue thus induced on the cells of the spinal ganglia; and Fish has examined the nerve cells after execution of a criminal by electricity, in which 1740 volts had been passed, and in which he found notable vacuolation of nerve cells. In a second case, however, as well as in animals killed by the electric current, he quite failed to find similar changes. All such attempts are too coarse to teach us anything with regard to normal physiological processes; and the appearances resulting can only be classed with pathological results, such as would ensue where chemical, thermal, or mechanical irritants were employed. Normal fatigue has been, however, studied in the ganglia and brain of the honey bee, the sparrow, and pigeon (*Hodges*). In such phases the nucleus lost its network or reticulated aspect, decreased in size, and became crenated in contour. The cytoplasm usually shrinks considerably, does not stain so readily, and loses its affinity for oxygen: occasionally vacuolation occurred. The normal condition was only *very slowly* restored after prolonged rest, five hours' stimulation requiring twenty-four hours' repose for complete repair. Gustav Mann describes the same results, affirms that there is a diffusion of chromatin within the nucleus, and that cell, nucleus, and nucleoli all alike increase in size in states of activity of the neuron. The chromophil granules in the cytoplasm show slight increase during functional activity, and diminution in amount and diffusion only with extreme fatigue (*Lugaro*).

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* Dr. Adolf Meyer has described several types of chromatolysis occurring in degenerative diseases of the neuron. *American Journ. of Insanity*, vol. liv., No. 2.

Lesion.—If by section, ligature, compression, chemical or thermal irritation the axon be destroyed, beyond the changes which are well-known to result in the peripheral segment and muscle to which it is supplied, we find the motor cells subject to a chromatolysis, such as has been already described—viz., primary increased size of cell, scattering and diminution of chromophilic granules from the centre outwards, wandering of the nucleus to the periphery without any change in its integrity, and, eventually, a lessening of the size of the cell. Such changes have been established by the observations of Nissl, Marinesco, Lugaro, and v. Gehuchten within 40 hours after lesion; and repair does not set in for two or three weeks, nor is the normal re-established for about three months.

In such cases no change occurs in the cytoreticulum; the achromatic constituent remains intact, whilst the complete repair undergone proves that no genuine *degeneration* has taken place, but that the process is one of simple deranged function. A much more rapid chromatolysis, followed by *destruction* of the cell, however, occurs, in the case of the spinal ganglion cells, in section of the peripheral nerves (*Marinesco*); a fact explained by their severance from all trophic influence.

Configuration.—Recent research into the general morphology and intimate histological structure of the nerve cell has so far enlarged the boundaries of our knowledge, that it becomes essential to start with a definite terminology which includes no ambiguous terms for the complicated apparatus presented as the modern conception of a nerve cell.

The body of the cell, including all its contents, we shall denominate the **cell** or **neurocyte**; the protoplasmic body of the cell, in contradistinction to the nucleus, may be termed the **neurosome** or **cytoplasm**. If the cell is provided with protoplasmic processes, we speak of the main trunk as the **dendron,** and the finer arborisations, as the **dendrites.** If one of these occupy a polar position whilst the others arise from the base or sides, as in the pyramidal cells of the cortex, we should designate the former as **apical** or **primary** dendrons, and the others as **lateral, basal,** or **secondary** dendrons. The dendron, be it remembered, is not an essential part of the cell, since many nerve cells have no dendron. The axis-cylinder may be much more conveniently termed the **axon,** which may be naked (**non-medullated**) or **medullated,** and such branches as arise along its course, as **collaterals.** The distal termination of an axon or its collaterals in a plexus, we speak of as a **terminal arborisation**; and for the whole system thus embraced, *i.e.*, the neurocyte with its axon, collaterals, terminal arborisations, and, if present, the dendritic expansions, we use preferably the term **neuron** in the original sense adopted by Waldeyer.*

The Neuron.—The nerve cell or neurocyte may vary much in contour; it may be pyramidal, small or large in size, giving origin to apical or primary dendron, whilst from its sides and base emerge secondary dendrons as well as the axon; it may assume a swollen, inflated, or flask-like configuration; or may be elongated in a long spindle-like element, either pole being the origin of extended processes, as seen in the lowermost layer of the cerebral cortex and the claustral formation. It may assume an irregular, angular, polyhedral form; or, again, may resemble a large granule, in which the nucleus is of large proportionate size, and the cell protoplasm greatly limited in amount, as is the case with the granule cells of the cortex cerebri, the cerebellum, and other regions of the nervous centres. It must be understood that our description of the contour of the neurocyte and the size of its individual parts, refers entirely to fresh frozen sections—the deforming influence of chrome reagents, and of the silver and mercurial reagents of Golgi and Cajal being so great as to require checking by examination of fresh specimens. It is not, however, upon these diversities in contour that we can lay such emphasis as was formerly done—but the whole neuron—cell, axon, and dendron (where present) must be considered in all its diversities of form before we can relegate the unit to its proper place in a rational classification. We shall, therefore, consider shortly the more important forms of neuron presented to us in the cortex cerebri, cerebellum, and their dependencies. The elements thus to be discussed are as follows:—

(1) Angular and sensory cells of Golgi.
(2) Granule cells (cerebrum, cerebellum, cornu, olfactory lobe, retina).
(3) Pyramidal cells of cortex and its varieties—
  (α) Motor cell.
  (β) Pyramids of cornu.
  (γ) Cells of Purkinje.
(4) Cells with ascending axons.
(5) Cells of peripheral zone.
(6) Mitral cells of olfactory bulbs.
(7) Inflated and irregularly globose cell.
(8) Spindle cells.

(1) **The angular cell** is of very irregular contour, occasionally approaching an oval, a pyriform, or even a fusiform outline. It quite as frequently assumes more of a pyramidal, and still more frequently an inverted pyramidal contour, due to a bicorned formation of its

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* *Deutsche med. Wochenschr.*, 1891.

Plate II.

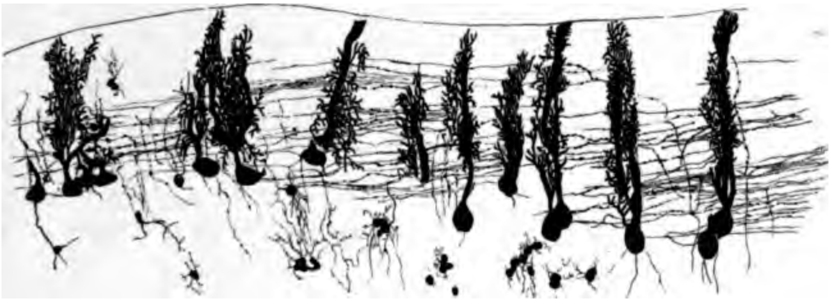
X 110.

FIG. 1.

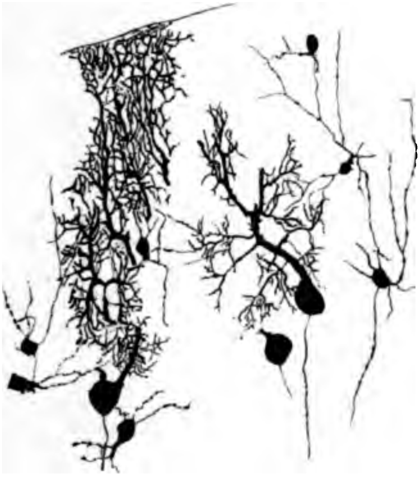
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FIG. 2.

CEREBELLAR CORTEX: CELLS OF PURKINJE (HUMAN).

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uppermost pole; in fact, its distinguishing feature is this great irregularity in form. These cells peculiarly characterise the **second layer** of the cortex, and may be well seen in the **great limbic lobe** of the Pig or of the Sheep. To see its more notable development, we must turn to the lower arc of the limbic lobe in the Rat or Rabbit, or what corresponds to the gyrus hippocampi. Here, within the area limited by the limbic sulcus (Plate i., A.T.C.), are seen dense clumps of these irregularly-shaped nerve cells closely appressed, usually measuring 18 μ × 10 μ in size, with a nucleus of 9 μ in diameter. An important character borne by these irregular elements is the relatively large size of the nucleus, as compared with the protoplasm of the cell; this feature, seen in these elements in the rodent, in the sheep, the pig, and other mammals, is also seen in the cortex of man.* In **osmatic mammals,** it forms, as we shall see, a special cortical type, and we are struck further by the dense meshwork of ramifications which arise from its outermost branches. The angular cell may be recognised at other levels, but it is here (gyrus hippocampi) that its richest development occurs.

Amongst these angular cells are scattered globose or shot-like cells —the "Cellule polygonale" of Cajal, which, as seen by the silver-chromate method, and especially by the modified Cox's process, are apparently identical with the sensory cells of Golgi. These cells throw off numerous protoplasmic processes, which are rough and thorny, like the dendrons of the pyramidal cells, and which divide and subdivide as they spread outwards from the cell. Moreover, from each cell arises an extremely delicate naked axis-cylinder or neuron, which throws off along a complicated course numerous offshoots or delicate collaterals, which conjointly form a complex system of curves around the cell or stretch into distant parts of the cortex, either upwards, downwards, or horizontally in its peripheral zone (fig. 9).

(2) **The granule cell** is a small element, averaging 10 μ × 8 μ in size, and many not larger than 9 μ, and with a nucleus of 4 μ to 5 μ in diameter. Slightly conical in form, with relatively large nucleus, the delicate protoplasm extends into several extremely fine processes: an apical process being also often present. This element forms an important constituent of sensory realms of the brain, and may be seen as a densely grouped formation in what we have elsewhere described as the **modified upper limbic type** in the rodent † (Plate vi.). In the histological study of the cortex, these two varieties of cell—the **angular** and the **granule**—are so diverse in forms, and their regional

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* "The Cortical Lamination of the Motor Area of the Brain," *Proc. Royal Soc.*, No. 185.

† "The Comparative Structure of the Brain in Rodents," *Philosoph. Trans.*, part ii., 1882, p. 709.

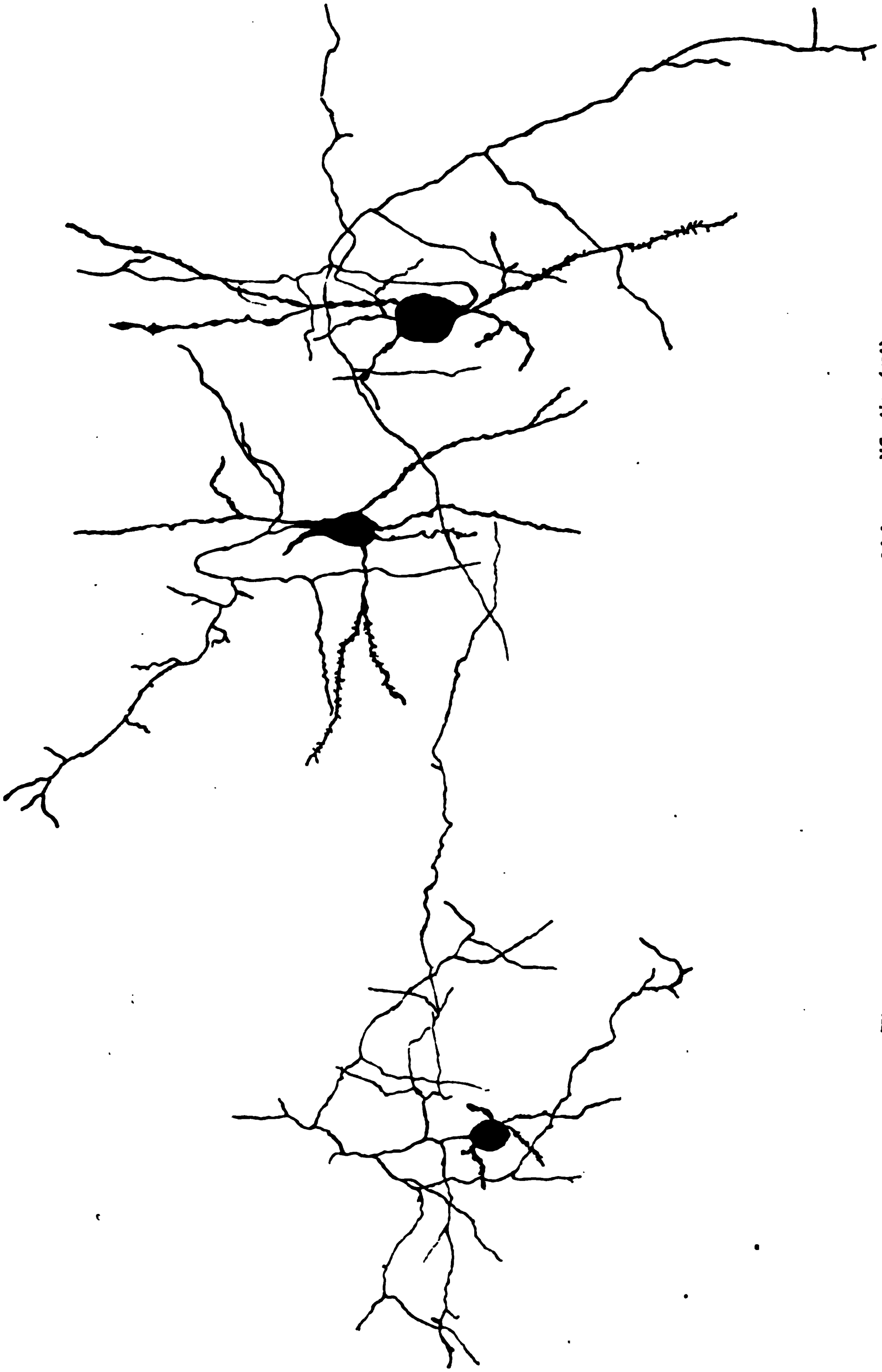

Fig. 9.—Cerebral cortex: nerve cells of second layer: higher amplification (rat).

distribution is so distinct, that it would be inexcusable to confuse the two formations as of identical constituents.

Several varieties of granule cells exist which, by the older methods of research, were not differentiated, but can now be readily distinguished. The more important are the granule cells of

(1) Cerebrum; (2) Cerebellum; (3) Cornu Ammonis; (4) Olfactory bulb; (5) Retina.

By some authors it is doubted if either of the last two is correctly to be claimed as a true nervous element; both are supposed to be devoid of that all-essential feature of the neuron—viz., an **axon.** Protoplasmic processes certainly do exist, branching similarly to those of other nervous elements; but, in the absence of the axon, the granule elements of the olfactory bulb and the spongioblasts of the retina might, by some, be justifiably classed as connective structures.* Those of the cerebral cortex average 10 μ by 8 μ in size, many not larger than 9 μ, with a nucleus of 4 μ to 5 μ in diameter. Slightly conical in form, with relatively large nucleus, the delicate protoplasm extends into several extremely fine processes; an apical process being also often present. This element forms an important constituent of **sensory realms** of the brain, and may be seen as a densely grouped formation in what we have elsewhere† described as the modified upper limbic type in the rodent (Plate iv., fig. 1).

The small granules of the cerebellum on the other hand, forming the densely packed inner cortical layer, give origin to three or four small stunted protoplasmic processes, rapidly ending by subdivision amongst the neighbouring granules. The axon arising from one of these same processes passes straight up into the pure grey layer, and, bifurcating at right angles, each division courses in contrary directions parallel to the cortical surface. Their intimate relationships we shall refer to later on (Plate viii., fig. 1).

In the fascia dentata of the cornu Ammonis the granule element is peculiar; its protoplasmic processes arise wholly from the outer pole of the cell to spread widely in a rich plume into the peripheral zone. Its axon passes through the granule layer, and, after giving off numerous collaterals, becomes continuous with the so-called "Moss-fibres" of this region, which ramify across the dendrons of the large pyramidal cells (Plate vii., fig. 2).

(3) **The Pyramidal Cell.**—From its uniform contour, large size, very general distribution (regional), and depth of formation, this cell has come to be regarded by many as pre-eminently *the* nerve element of the cortex.

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* See "Notes on Granules," by Alex Hill, M.D., *Brain*, vol. xx., p. 125.

† "The Comparative Structure of the Brain in Rodents," *Philosoph. Trans.*, part ii., 1882.

Pyramidal is a name appropriate only to those cells which have undergone the corrugating effects of chrome, other hardening reagents, or desiccation. In the fresh state they are wholly different in configuration from those seen in hardened specimens. On the other hand, Meynert is far too exclusive in stating that their true form is that of a spindle; in fact, they are very variable in form, often plump and rounded off at their base, lengthened out and attenuated at their apex. The pyriform contour is very general—minute angular projections of protoplasm on all sides mapping out the origin of delicate processes. Occasionally they are elongated and truly fusiform, especially in certain definite regions of the cortex; and yet others occur where the body of the cell is larger above than below, its lower end being in fact attenuated, so that the cell has the contour of an inverted ovoid. Where they approach the pyramidal form it is usually one of a very irregular triangle, with sides irregularly broken by numerous dentations caused by the processes distributed therefrom. From the summit of the cell arises the **apex process,** or primary dendron, directed radially to the surface of the cortex, whilst on either side from the basal aspect—the secondary—fairly stout branches diverge—not at right angles, but forming an obtuse angle (of about 120° very uniformly) with the long axis of the cell. It appears to us that these stout lateral branches (which, with the apical, form by far the most prominent extensions of the cell) explain its triangular or pyramidal form upon shrinking in chrome fluids. The apex process, or main dendron, passes upwards towards the outermost layer of the cortex, to which it usually, but not invariably, extends; small branches arise from thickened axils along its course, and, eventually, the main branch divides and subdivides into a large number of branches forming a rich plume within the first cortical layer. These dendrites appear to terminate immediately beneath the pia, or sweep horizontally beneath the latter for varying distances. The most important feature revealed by the silver-chrome method as regards this apex process and its dendritic plumule, is the fact that the branches are *apparently terminal*, and are clothed throughout their course by minute thorn-like appendages, each surmounted by a minute bulb or bead-like head, in the intervals between which lie the axons or axis-cylinder processes and the terminal arborisations formed by the latter (Plate xiii., fig. 2). Cajal* terms the primary dendron the **trunk** (*tige*) as being the earliest evolved of the protoplasmic processes. These bodies, therefore, throw off three sets of protoplasmic fibres:—

(*a*) The **apical dendrons,** which are by far the most conspicuous, and always radiate to the surface of the cortex;

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* See on this point—Cajal, "Sur la Structure de l'Ecorce Cérébrale," *La Cellule*, 1891, tome vii., p. 135. Also the author on "The Structure of the First Layer of the Cortex," *Edin. Med. Journ.*, June, 1897.

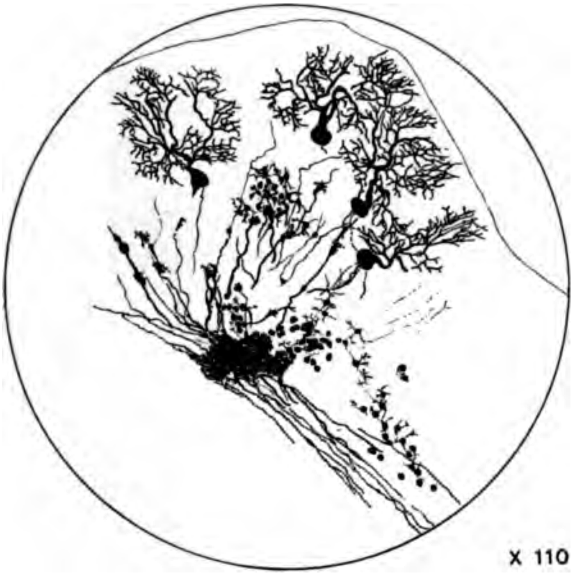

FIG. 1.

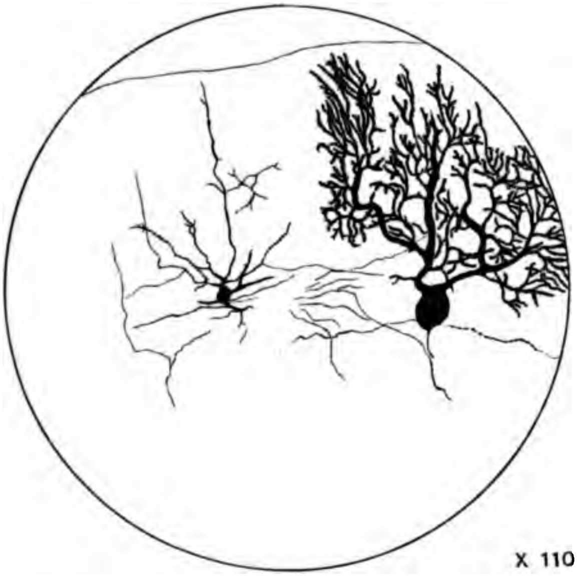

FIG. 2.

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(*b*) The large **basal dendrons,** running obliquely outwards and downwards on both sides; and

(*c*) Numerous very delicate lateral dendrons radiating from all intervening districts of the surface into the nervous meshwork around.

These lateral protoplasmic processes give origin to numerous branches which ramify extensively in the neighbourhood around the cell, and, like those of the apical plumules, are also invested by thorny appendages (*épines*).

The axon passes downwards from the lower pole of the cell, or from a basal dendron here, to enter the white medulla of the corona radiata: its constitution and relationships can be better relegated to our description of the first variety of the pyramidal cell, viz.—the Motor.

Each cell contains an oval **nucleus,** with well-defined **nucleolus,** 7 μ × 5 μ in size. The dimensions of the cell vary from 12 μ × 8 μ, in the more superficial to 41 μ × 23 μ in the deeper layers.

(*α*) **The Motor Cell.**—We are alive to the exception which may be taken to any such implication as the above designation conveys; yet, as it appears to us that the argument in favour of their motor endowments has been materially strengthened by further examination of the question, we prefer this designation to that of **giant pyramids,** which was proposed by Betz, more especially since these elements may be recognised by certain features in certain regions, where they by no means deserve the ephithet "*giant cell,*" being even smaller than the lower cells of the third layer above them. The motor cell, taking into consideration the more characteristic elements, are the largest cells found in the cerebral cortex. Some of the largest of these measure 126μ in length by 55μ in the shorter diameter; the average dimensions of a very large number in the ascending frontal convolution being 60 μ × 25 μ. The extremes are 30 μ and 96 μ for length, 12 μ and 45 μ for breadth. They contain an oval nucleus, 13-20μ in greater by 9-12μ in lesser diameter. In form these cells are very variable, usually much swollen, plump-looking bodies; they are elongated and attenuated towards their apex process, throwing off the greater number of protoplasmic processes from near the opposite pole. The contour of these nerve cells appears related to the number and size of their branches—*i.e.*, the greater the number of such processes, the more irregular the contour; whilst the apical and basal processes being usually the larger, the cells tend to lengthen out in their direction and assume a more or less fusiform outline. Large processes, however, given out from various other points of the cell, greatly modify this spindle form, so that extreme variations in configuration occur. We shall see that we have reasons for believing that the primitive form of all these nerve cells is globose or slightly pyriform; that the fusiform outline is the next stage of their development; and that further modifications occur as other processes beyond the apical and basal extend laterally. So likewise we shall see *the reverse change undergone by the cell in the dissolutions of disease.*

The cell has, in the normal state, no **cell-wall**; but the appearance of such is readily induced by reagents and disease. In fresh specimens obtained from frozen brain, the cell is seen to consist of a delicate protoplasm, which appears to be directly continuous with its various processes; nor can any trace of the fibrillated structure of the cell-contents described by Max Schultze be detected unless, indeed, reagents be employed. Then we obtain, as by Nissl's method of staining, evidence of a complicated structure, to which we have previously referred (see p. 67).

The lower pole of the cell is usually **pigmented** as a normal condition, just as is the case with the multipolar cells of the spinal cord. A large round or oval nucleus enclosing a nucleolus is always present in these cells. Each cell throws off what may be termed **primary** and **secondary** protoplasmic branches—the former the apical extension; the latter including all other processes except the axon, whether coarse or delicate fibres. The division usually adopted by Continental writers is into centripetal (apical and lateral secondary processes) and centrifugal (axis-cylinder or axon).

The **apex process** or primary dendron, formed by the gradual attenuation of the cell, passes straight up through the superjacent layer of cells, throwing off in its course several collateral protoplasmic processes, which usually run obliquely upwards and outwards from the trunk and lose themselves in the neighbourhood around; whilst, as in the pyramidal cell above described, the main trunk, on entering the peripheral zone, furnishes a rich dendritic plume to that layer, the fibres of which similarly present a fine hispid appearance from minute projecting spines. Since in the motor cortex these nerve elements are aggregated into groups or clusters (Plate v.), these apical processes, closely approximated, often run in sheaves through the more superficial layers. A basal **protoplasmic process** often arises in like manner from a gradual attenuation of the opposite pole, as in fusiform cells, and large lateral branches may strike out from this extended pole. The basal process usually, however, continues downwards for some distance, when it gains a thin investing sheath of medulla, which gradually thickens upon it, converting it into a true medullated nerve fibre. Hence this process is termed the **axon** or **axis-cylinder process.** Upon this Meynert remarks—"It is the more rarely seen because, being the process which enters the medulla, its direction is dependent upon the angles formed by the fasciculi of the latter, which by no means form a straight line with the apical

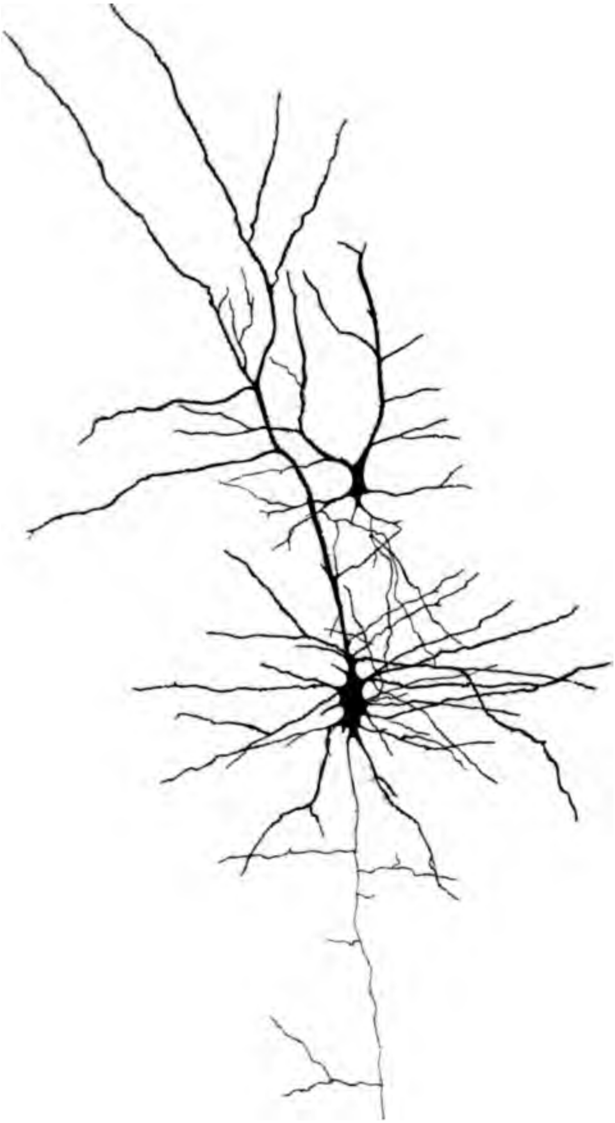

Fig. 10.

processes of the pyramid."* This obliquity of position, therefore, necessitates its being cut off in sections on a plane with the radiating apical processes. This statement, however, no longer remains true for the *new* methods of preparation, since the wealth of structure and comparative thickness of sections examined in silver-chrome preparations admit of the axon being readily seen in most cells (fig. 10).

The **secondary** or **lateral processes** which radiate from the cell on all sides, unlike the primary, divide and subdivide almost immediately after their origin, and interlace in the intricate webwork of nerve and connective fibre around the cell. Schaffer has recently indicated that these secondary processes are readily distinguished from the axons by Nissl's method of staining; spindle-shaped chromophil bodies stained by methylene blue are grouped around these secondary processes, but the axon is devoid of such bodies.† We are apt to overlook the extreme complexity of structure in vertical sections of the cortex, and should compare with such sections others carried *across the long axis* of the cell (obtained by placing the cortex surface downwards on the freezing microtome, and cutting down to the level of these cell groups). Such sections show us one or two cells as the centre of an area to which their branches are distributed; their finest ramifications crossing and recrossing, *but not inosculating*, with those from adjacent cell territories. The termination of such branches is regarded as absolutely free—*i.e.*, no true meshwork by *re-union* of the branches of one cell with the other is supposed to occur; at least, such is the appearance presented by sections prepared by the Golgi and Cox methods of examination.

We have observed as many as eighteen main processes diverge from a single cell in such sections; in vertical sections the average number seen is about seven, but as many as fifteen have been observed. When it is remembered that no single section can show (as the teazing methods, however, do) the actual number of branches in any single cell, the above statements will indicate the wealth of communicating branches which these "motor units" possess.

The axon, as before stated, arises directly from the gradual attenuation of the lower pole, or it may arise indirectly from a basal dendron. It passes downwards towards the medulla, throwing off at right angles along this course some six to ten extremely fine **collateral** branches (*Cajal*), which end in delicate terminal ramifications around. The axon still maintains the same uniform dimensions, and, entering the medulla, it either bends at right angles to continue its further course or bifurcates into two divisions running in opposite directions.

When we come to examine what appear to be the corresponding cells in the cortex cerebri of some of the lower mammals, we find certain strong points of resemblance, together with certain distinctive features by which we may very readily recognise them as not human. Thus in the **pig,** in lieu of the great irregularity in marginal contour seen in man, we observe, on the contrary, a notable uniformity of contour, the elongate pyramid being the almost universal form. "They resemble closely, both in size and form, the large pyramidal cells at the *deepest portion* of the third layer in bimana, quadrumana, and the large carnivora, as also the *ganglionic cells* in the parietal and temporo-sphenoidal lobes of man. Nowhere do we find the irregular, swollen,

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* *The Brain of Mammals*, Stricker's Handbook, p. 387.

† *Neurologisches Centralblatt*, Dec. 15, 1893.

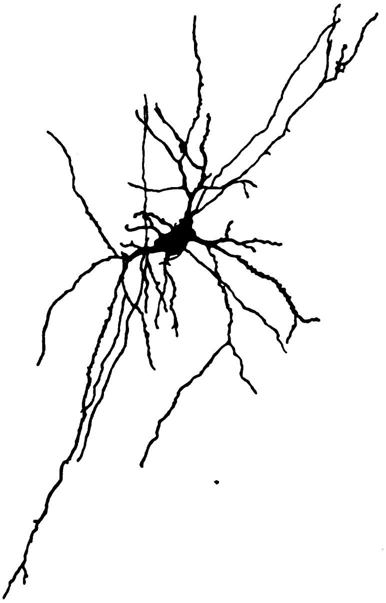

Fig. 11.—Cerebral cortex: nerve cell from deeper zones of cortex (human).

and at times almost globose cells so frequent in the motor area of the human brain."* Again, in the **sheep,** we fail to find the plump rounded cells of man and the higher mammals; but the cell is more variable in form than in the pig, the elongated pyramid being interspersed freely with long spindle forms and large numbers of a peculiar "**horned**" **cell,** in which the apex process is bifurcate at its origin near the cell. They measure on an average 46 μ × 11 μ. In the **cat,** however, these elements are plump, oval, and pyriform; average 51 μ × 21 μ in size, with an occasional gigantic cell of 106 μ × 32 μ;

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* "Researches on the Comparative Structure of the Cortex Cerebri," *Proc. Roy. Soc.*, part i., 1880.

and are grouped together in well-marked clusters. In the **rodent** (rat, rabbit) the type of cell approaches that found in the sheep and pig.

One may readily perceive the remarkable resemblance between these cells and those of the anterior cornu of the cord in chrome-hardened preparations, but still closer appear their affinities in structural arrangements when teazed-out specimens of brain and cord are compared. The inference that these cells are specialised elements rests on this resemblanee, on their exceptionally large size and abrupt commencement, and the peculiar clustered groupings assumed in a region which has been shown by Ferrier to possess motor endowments (see p. 121). Meynert, on the other hand, who fails to recognise these larger cells,* draws a parallel between the whole of the pyramidal cells of the third layer and the motor cells of the cord. His statement is as follows:—"If we remember that the anterior roots of the spinal cord, at their origin in the anterior cornua, are connected with elements which, through the slenderness of their bodies, the gradual transition of these bodies into the protoplasm of the processes, and the greater number and size of the latter, are sharply differentiated from the cells in which the posterior roots originate in the interspinal ganglia, these being tumid and provided with few and attenuated processes, an affinity in point of form is at once seen between the pyramids of the cortex and the former, which is common also to the cells of origin of all motor cerebral nerves, and permits an analogy to be drawn in regard to the significance of the pyramids of the cortex." †

(β) **Pyramids of Cornu.**—The great pyramidal cells of the cornu Ammonis in close approximation to the fimbria are peculiar in several respects. From their superior or apical pole arises a very thick dendron, which almost immediately breaks up into a coarse dendritic arborisation—stunted, and in nowise resembling the finer dendritic plumules of the pyramids of the cerebral cortex. Again, the collaterals arising from the axons of these cells pursue a notably *recurrent* course, passing by their cells to ramify eventually over the dendrites of the smaller pyramidal cells of the cornu. These smaller pyramids, on the other hand, more closely resemble those of the cortex cerebri; they are usually pyriform or spindle-shaped, or, far less frequently, pyramidal in contour (Plate vii., fig. 2).

(γ) **Cells of Purkinje.**—The flask-like cells of the cerebellar cortex were never satisfactorily displayed in chrome preparations by the older methods of staining with carmine, hæmatoxylin, &c., the profuse branchings into the peripheral layer were well revealed by aniline blue-black (*Sankey*); but yet their axons were revealed merely by a very short projection from their base, their continuation being lost in the subjacent granule layer. Moreover, the elaborate basket-work found around the cell, as well as the plexus of branches from underlying cells, which spread over their main dendrons, were not discovered until the silver-chrome method of Golgi came to our assistance. By this means the cell appears as a flask-like element throwing upwards one or two main dendrons, which divide and subdivide into a rich dendritic arborisation as far as the pial surface; whilst the axon arising from its base passes through the granule layer and, throwing off several collaterals, is eventually lost in the medullated tract of the lamina. Their relationship to the basket-work and plexuses around their dendrons will be more fully described in the section on the cerebellar cortex.

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* See the diagram of the five-layer type in Meynert's *Psychiatry*, and also in *Sydenham Society's Trans.* of his monograph, fig. 234.

† *Brain of Mammals*, p. 387.

(4) **Cells with ascending Axons.**—Certain cells scattered through the pyramidal and ganglionic layers of the cerebral cortex have been

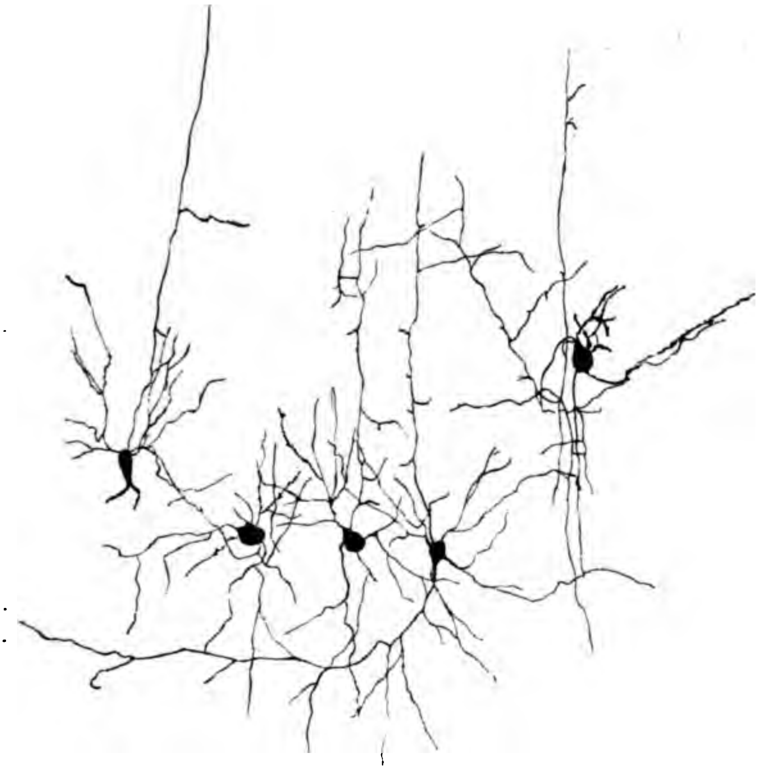


Fig. 12.—Cerebral cortex: nerve cells of second layer with extensive ramification of axons (rat).

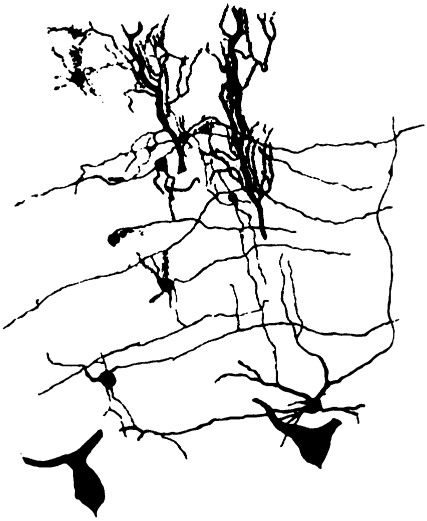
lately shown by Martinotti to have the usual distribution of branches reversed. As frequently spindle-shaped as triangular and throwing off protoplasmic processes both ascending and descending, the axon, on the other hand, arising often from a coarse apical dendron, *ascends* to the peripheral zone. Here it breaks into two or three branches which take a horizontal course, and subdivide in a very extensive terminal ramification in this the lowermost region of the outer cortical layer. Cajal describes a similar arrangement of certain such ascending axons immediately beneath the second layer (fig. 12).

(5) **Cells of the Peripheral Zone.**—These nervous elements, to which great importance has lately been assigned by Cajal and others, are either fusiform or stellate; the former being bipolar and throwing off from either pole a horizontally disposed protoplasmic process which eventually thins off into several genuine **axons,** disposed parallel to the surface; whilst exceedingly delicate collaterals arise from these to ramify upwards in the peripheral zone. In like manner, the stellate cells are multipolar, yet their branches are disposed along horizontal planes, and throw upwards collaterals in the same manner, all of which have the aspect of delicate axons. Here, then, it will be observed we have cells provided with no genuine dendritic expansions, the protoplasmic processes being directly continuous with the axons arising therefrom; moreover, the cells never trespass beyond the boundary of the cortical zone in which they lie.

(6) **Mitral Cells of the Olfactory Bulb.**—Another very peculiar type of cell is illustrated by the so-called "mitre" cell. This element is triangular, or more swollen or globose in contour, giving origin to lateral ramifying processes, and to a stout dendron, which, arising from its superficial aspect, passes downwards to an olfactory glomerulus, and breaks up within it into a rich terminal plexus of branches. An axon also arises from the deeper aspect of the cell to become continuous with an olfactory nerve fibre (Plate iv., fig. 2).

(7) **The Inflated or Irregularly Globose Cell.**—The nerve cell to which the epithet **inflated** has been given has not been, so far as we are aware, described amongst the constituents of the cerebral cortex by any former writers on the subject. We first drew attention to it as a specialised cell, forming a distinct layer of the cortex, in a Memoir on the Comparative Structure of the Brain in Rodents (1882), and subsequent examinations fully confirm the description then given. The brain of the mole, rat, or rabbit is especially suitable for demonstrating the presence of this element. The cell which occupies the position of the small "pyramidal" and angular bodies of the second layer is no longer of pyramidal form, but swollen, inflated, globose, or flask-shaped, and, moreover, of *far greater size.* The average dimensions attained by it are $37\,\mu \times 32\,\mu$, with a nucleus of $13\,\mu$; some are more

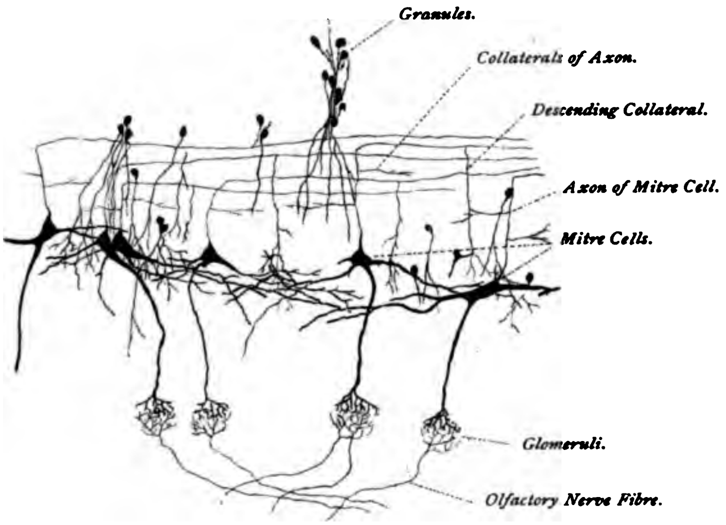
Plate IV.



X 110.

FIG. 1.

CEREBELLAR CORTEX: STELLATE NERVE-CELL NEAR CELLS OF PURKINJE (HUMAN).



X 110.

FIG. 2.

OLFACTORY BULB OF RAT: SEMI-DIAGRAM.

Bale, Sons & Danielsson, Ltd., Lith.

elongate, measuring 46 μ × 27 μ. Hence, these elements are *more than double* the size of those usual to this position, and exhibit the apparent anomaly of *large cells* in the cortex *superimposed on* a layer of *small pyramids.* The region in which they are found is really the hindmost extension of the lower limbic lobe (modified lower limbic type) in the rodent.

As will be seen later on, the second layer of the cortex in the lower limbic arc is characterised by its peculiar closely appressed clusters of small pyramidal or angular elements, with bifurcate apices, which subdivide into a dense meshwork of fibres; farther back, in the region above indicated, these elements appear transformed into the inflated cell, retaining, however, their bifurcate apices and plexiform branching. The cell throws off numerous fine processes on all sides; its protoplasm—exceedingly delicate—shrinks greatly under the influence of alcoholic and other corrugating reagents, and should, therefore, be always examined in the fresh state. When acted upon by chrome it loses its characteristic appearance and resembles the vesicular cell, which, in the medulla and spinal cord, is regarded as possessing sensory endowments. It appears to us that the whole belt of the second layer of the cortex, out of which this specialised cell is developed, may subserve the same purpose—that of sensation in its various phases: the evidence on this point had better be considered at a later stage of our enquiries.

(8) **The Spindle Cell.**—This undoubtedly is also a specialised element. The cell is a narrow fusiform body, attaining the average dimensions of 25 μ × 9 μ, the largest being 32 μ × 13 μ, with an oval or fusiform nucleus 11 μ to 13 μ in length × 6 μ to 9 μ in breadth.* Their two principal branches arise from either pole so as to give them in many cases the aspect of bipolar cells; but, as indicated by Meynert, lateral projections also arise from these bodies.† Frequently this lateral branch becomes large, and the resulting angular projection of the cell-protoplasm into it gives the cell a triangular or triradiate form. The cell is regarded as an intercalated element of the connecting system of the brain, and since the claustrum is entirely composed of such elements, the term **claustral formation** has been proposed for it by Meynert. These elements are peculiarly prone to a nuclear proliferation, which occasionally accumulates into little heaps almost concealing these cells from view. In position they underlie the other layers of the cortex throughout its whole extent; whatever be the type of lamination, the lowest stratum will always present us with these spindle cells of the association system of the brain; this applies equally to the mammalian brain in general.

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* *Transactions of the Roy. Soc.*, 1882, part ii., pp. 714-15.

† *Op. cit.*, p. 389.

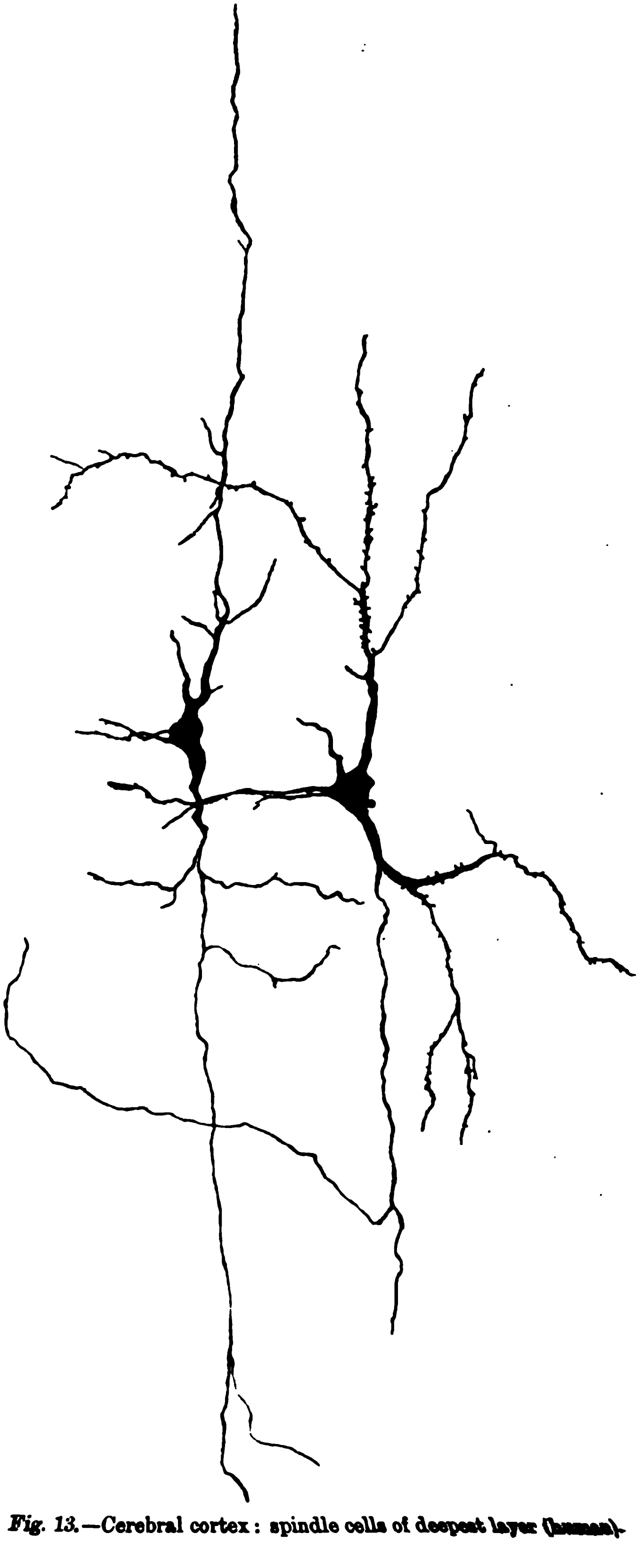

Fig. 13.—Cerebral cortex : spindle cells of deepest layer (human).

We have elsewhere indicated the existence of a perfectly **globose cell**—with a single delicate apex process, and two or more extremely attenuated processes—without any angular projections from the cell, but a perfectly uniform rounded contour—as existing normally in the second and third layers of the cortex of the ape, and as being specially characterised by this swollen globose contour, and great paucity of branches. They are met with in man only in forms of developmental arrest—in idiotcy and imbecility; but elements which remind us of these cells, occur in the second layer of the cortex of the pig. These may be early stages in the development of the more advanced forms of cortical cells, and may or may not have affinities to the inflated irregularly globose elements already described in a specialised cortex of the rodent.

The above constitute the various forms of nerve cell which occur in the mammalian cortex, and we must now direct attention to its other histological constituents: these consist of—

(*a*) Nerve fibres;
(*b*) Blood-vessels;
(*c*) Connective matrix or neuroglia;
(*d*) Lymph channels.

(*a*) **Nerve Fibres.**—As is well known, nerve fibres, central and peripheral, present varied forms, corresponding to five stages of development—from the ultimate fibril up to the ensheathed and medullated fibre of the peripheral nerve trunks. The last, the most perfect and complex form, does not occur in the nerve centres at all. In the cortex, as well as the nervous centres generally, three forms of fibre are met with—(1) The primitive fibril. (2) Naked, non-medullated or protoplasmic process. (3) The medullated fibre devoid of a sheath of Schwann.

(1) **The Primitive Fibril.**—The representative of the ultimate divisions of the non-medullated fibre, is an excessively delicate attenuated thread, revealed only by an amplification of 500 diameters; and which, as the result of *post-mortem* change, becomes beaded or shows varicosities along its length. They are observed readily from the occurrence of this change by lower powers of the microscope (× 350), especially by imbibition of fluid around, which causes them to swell up into large oval varicosities. Such delicate beaded threads are seen at all depths of the cortex in fresh sections obtained from frozen brain, treated with osmic acid, ·25 per cent., and protected by a cover-glass; but they are also traceable in sections which have been hardened by chrome, especially in the lowermost layers. They are seen in many cases to arise from the subdivision of larger fibres; they are perfectly homogeneous, betraying no internal structure to the highest powers of the microscope.

(2) **The Naked or Non-Medullated Fibres,** the **protoplasmic processes** of Deiters, form an important constituent of the cortex. From what has already been stated respecting the mode of branching of the nerve cells, it will be apparent that the protoplasmic extensions occur in complicated meshworks throughout the cortex. The apical process and lateral extensions pass by subdivision into an intricate meshwork of fibrils: the basal process becomes invested lower down with a protecting layer of medulla. This is the process to which, properly, should be restricted the term **axis-cylinder process**; and, for all other extensions of the cell protoplasm, the term **non-medullated fibre** or **protoplasmic process** should be applied. Since, however, recent research has demonstrated the fundamental distinction betwixt the protoplasmic processes and the axis-cylinder (the former being invested by spiny projections, and becoming more and more attenuated as they subdivide; the latter invariably smoother, and retaining a uniform diameter in spite of numerous collaterals which emerge from it) it would be well to adopt the terms of Dendron and Axon as applicable to these two. Eventually the axon may terminate as a rich arborisation around other cells. Such processes are very variable in size; but, at their origin from the various cells of the cortex, they range between 1 μ and 6 μ in diameter: the lateral processes in particular become rapidly attenuated by subdivision, but yet may be occasionally traced over very lengthy tracts: the apex processes, running to the uppermost layer of the cortex, may often be traced to their termination here.

These fibres exhibit, under certain conditions of examination, a linear longitudinal marking, which has been described as "fibrillation" by certain authorities (*Max Schultze,** *Landois and Stirling* †): the homogeneous nature of the non-medullated fibre and axis-cylinder has, on the other hand, been maintained by *Kölliker,*‡ *Waldeyer,*§ and others. Since those who support the view of the fibrillation of the axis-cylinder regard the fibre as a compound of the ultimate fibrils already described, separated by a small quantity of interfibrillar substance, and believe them to be continuous through the ganglion cell in what they describe as a well-marked fibrillation of its interior, the question of the homogeneity or of the fibrillated constitution of the axis-cylinder becomes of fundamental importance in neurology. Such fibrils would be regarded as isolated tracts of conduction throughout their length, the nerve fibre itself being a far more complex structure than what it was once regarded as being, and the cell itself would have a far different significance. Nor, according to some, need this *visible continuity* of the fibrillæ be demanded to establish the case—more or less fusion may occur throughout the length of the fibre; and the splitting up into fibrillæ only be observed at the centric and peripheric terminations as an indication of the fibrillar constitution of the axis-cylinder and its lines of molecular disturbances. If these protoplasmic processes and axis-cylinders be submitted to the action of silver nitrate in the dark, subsequent exposure shows them to be marked by a peculiar transverse striation, first indicated by Fromann.* Their significance is unknown.

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* Stricker's *Human and Comparative Histology*, Syd. Soc., p. 158.

† *Text-Book of Human Physiology*, vol. ii., p. 768.

‡ *Gewebelehre*, 5th Aufl., 1867, p. 244.

§ *Zeitschrift für Rationelle Medicin*, Band xx., 1863.

(3) **Medullated Fibre or Axis-cylinder Process.**—This may be either examined in the radiating expansion arising from the medullated core of a convolution at the site of the spindle layer of cells; or, in the different intracortical arciform belts found at a higher level. The medullated fibre of brain and spinal cord consists simply of an axis-cylinder with an investing **sheath** of **myelin,** which gives to the medullated fibre its white appearance, non-medullated fibres having a grey translucency. The myelin is of fluid consistence, and appears limited simply by a very friable, soft, protoplasmic envelope (*Cornil* and *Ranvier*†), and not by the strong resisting **sheath (of Schwann)** which invests the *peripheral* fibres. Kühne and Ewald have proved, by the use of trypsin, that the axis-cylinder is enclosed in a sheath of indigestible horny material, which they term the **keratoid sheath.** In the peripheral nerves, however, this keratoid sheath not only embraces the axis-cylinder, but, being reflected on the inner aspect of the sheath of Schwann, really serves to enclose the white medullated substance or myelin. In these more complex peripheral fibres (to which we must divert, for the time, our attention), although the axis-cylinder is continuous throughout, the medullary sheath is not so, but presents at regular intervals annular constrictions named **Ranvier's nodes,** after their discoverer. Ranvier called the individual parts formed by these constrictions **interannular segments;** and showed that, whilst covered externally by the resistant structureless sheath of Schwann, both were interrupted at these constrictions. In a depression of the myelin, and between it and the sheath of Schwann, are the **nerve corpuscles**—one for each segment, consisting of an oval nucleus surrounded by a little protoplasm.

The neuro-keratin sheath, spoken of above, lies therefore on the axis-cylinder, and, reflected at each constriction upon the sheath of Schwann, enjoys the same segmentation as the other constituents of the nerve fibre. Traversing the medullated substance from the inner to the outer portion of the keratin sheath, are numerous transverse and oblique dissepiments, also of a horny nature, supporting the myelin (*Lantermann*). At the annular constrictions, there exists a certain amount of cementing material, which, when the fibres are treated with silver nitrate, becomes darkened, and appears as a small cross at these nodal points along the fibres. The silver penetrating at these nodes stains also the axis-cylinder to a very limited extent, producing **Fromann's lines.** It is at this site that nutritive fluids gain access to the axis-cylinder, which otherwise could not be reached through the keratin sheath and medulla. Here also staining reagents gain admission, and colour the axis; and the myelin, after imbibition of fluid by the fibre, exudes at these constricting rings, pressed out by the swollen axis-cylinder in the form of droplets, easily recognised by their spherical form and double contour.

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* Virchow's *Archiv*, Band xxxi.

† *Pathological Histology*, vol. i., p. 33. Trans. by A. M. Hart.

The medullated fibre of the central nervous system, however, possesses, as we have already remarked, no sheath of Schwann; it is consequently devoid of the constrictions or nodes of Ranvier, has no interannular segments, no nuclei along its length, nor does it exhibit any signs of Ranvier's cross on treatment by silver nitrate. The constitution of these centric medullated fibres, therefore, leads to a *more perishable* nature. They are *far less resistant* than those of the peripheral nerves, break up more readily into myelin spheres, or become extensively *varicose*. Hence, also, we find it difficult to stain such medullated fibres in fresh brain. The protoplasmic extensions take up aniline dye readily, becoming stained of a deep blue-black; but, where the medullated sheath intervenes, the reagent fails to penetrate except along a short length just beyond the first appearance of the sheath. This want of permeability is compensated for, as before stated, in peripheral fibres by the presence of the constrictions of Ranvier.

To stain the axis-cylinder throughout its length in these centric fibres, we must first displace the myelin.* This can be effected by prolonged immersion of the section in water, and subsequent staining with **aniline blue-black.** Twelve hours' immersion usually suffices to remove the whole of the medulla around the axis-cylinders; and the latter are then seen as slightly wavy, swollen bands, often strap-shaped, and occasionally contorted, from the alteration undergone by aqueous immersion. They all run from the cortex downwards into the core of the medulla, to which they converge in large numbers—deeply stained; and forming a striking contrast to the unstained aspect of nerve elements at this site in sections which have been prepared in the usual manner. But although such axis-cylinders present difficulties in staining along their length, they are well seen in sections across their axis: such cross-sections appearing, especially in the lowest layers of the cortex (spindle-cell layer), as a central dark axis (often slightly drawn out into a short filament), surrounded by a sheath of white medulla retaining its circular outline—the myelin having been apparently "fixed" by the osmium treatment. The medulla in these cases is not perfectly homogeneous, but has undergone a change which gives it a *frosted* vitreous aspect, with a very slightly granular appearance, the diameter of the fibre being from three to four times that of the axis-cylinder.* Large medullated fibres occur at this site, in section, measuring 13 μ across, with an axis-cylinder of 4 μ; but extremely minute fibres are seen intermingled with these larger forms also, if the field be carefully searched. As we shall see later on, certain morbid conditions of the cerebral cortex modify to a considerable extent the character of this investing medulla.

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* In the very minute medullated fibres of the cortex we have an exception to this rule—the axis-cylinder staining fairly well without displacement of its investing myelin sheath: a result due undoubtedly to the small calibre of the latter allowing a certain amount of permeability.

(*b*) **Blood-vessels of the Cortex**—(1) **Arteries.**—These vessels, as they dip into the cortex, vary in dimensions from 4 μ to 12 μ. They possess the three tunics which are recognisable to the naked eye in large arteries elsewhere, the *tunica adventitia*, *media*, and *intima*; but, as in these larger vessels microscopic examination reveals the fact that each of these tunics is separable into several differently-constituted layers, so the larger cortical blood-vessels exhibit in the innermost coat a double layer—an elastic and an endothelial layer.

The **intima**, or lining membrane of the artery, in the *fresh* state appears as a structureless membranous tube, with numerous oval nuclei, well seen in carmine-stained preparations, scattered over its surface. These nuclear elements are disposed longitudinally, *i.e.*, in the direction of the vessel's length. The action of a solution of silver nitrate ($\frac{1}{4}$ per cent.) reveals the fact that this tunic is not a homogeneous tube, but that it is constituted of large squamous endothelial cells, which look like polygonal flattened scales, united to each other at their margin by a cementing material, which is mapped out in black lines by its reduction of the silver salt. Moreover, it is then seen that the oval carmine-stained elements are nuclei of these flattened cells. The inner **elastic tunic** is, in the smallest vessels, a structureless membrane, seen as a bright wavy division between the endothelial and muscular coat in transverse sections of the vessel; in the larger arteries it is a distinctly fenestrated membrane, the representative of Henle's fenestrated and elastic laminæ, which can be stripped off in shreds from great arterial trunks like the carotid and axillary when they tend to curl at the edge and roll themselves up. It forms an important line of demarcation between the innermost and the muscular layer.

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* It must be borne in mind that there exists a certain definite relationship betwixt diameter of axis-cylinder and medullated sheath: the larger axis-cylinder always having a larger sheath and *vice versâ*.

The **tunica muscularis** or **media** consists of smooth or unstriped muscular fibre with oval or strap-shaped nuclei. Such fibres being arranged transversely to the long axis of the vessel, or, rather, coiling spirally around it, appear at right angles to the longitudinally-disposed nuclei of the *intima*. Where this tunic is well developed, a longitudinal section of the vessel will often show these muscle fibres arranged in series along the margin of the tube, their nucleus, also divided transversely, giving them the aspect of round nucleated cells. The limiting wall externally is also often thrown into slight wavy outline from the projection of these muscular fibres. In transverse sections of the small arteries one or two such muscle cells surround the open lumen. The muscular element does not enter largely into the constitution of the cortical blood-vessels. These vessels, like those of the cranial cavity generally, as well as those of the vertebral canal, have much thinner tunics than vessels of corresponding calibre elsewhere from this poverty in muscular elements and adventitial tunic (*Sharpey*).

The **tunica adventitia,** which in the larger arteries is a connective sheath directly continuous with the pia mater (*intima pia*), becomes in the smaller vessels an extremely delicate membranous investment, faintly striated or structureless, upon which are found connective corpuscles, the nuclei of which are round or somewhat oval. A membranous nucleated tunica adventitia, similar to the above, can be readily observed in larger capillaries of the hyaloid membrane of the frog (*Eberth**). The corpuscles in this adventitial sheath form a very delicate protoplasmic structure, of fusiform or stellate outline, shrinking notably with hardening reagents and desiccation of fresh brain, so as to bring their nucleus much more prominently into view; in fact, mounted specimens usually show the nuclei only along the course of the adventitial coat. As we shall see later on, these nuclei are prone to extreme degrees of proliferation. Closely applied to the tunica media, as a rule, this adventitial sheath is in certain conditions widely separated from the vessel's wall in ampullar dilatations, and at all times leaves a space between it and the middle coat in the angle formed by the bifurcation of the vessel. The latter, with its sheath, traverses channels in the cortical substance which form a wall limiting the distension of the vessel. This limiting channel has no definite endothelial lining, so far as can be discovered by the silver treatment;

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* See Stricker's *Human and Comparative Histology*, vol. i., p. 287, fig. 53.

it is termed the **perivascular channel** of His, and is continuous with the **epicerebral space** between the **intima pia** and the outer surface of the cortex. Traversing this perivascular space are numerous delicate fibrillar processes, which, arising from stellate cells in the substance of the cortex, thus form connections with the adventitial sheath of the artery.

(2) **The Capillaries.**—These channels of intercommunication between artery and vein are of extremely fine calibre in the cortex. Taking the capillaries of all regions, excepting the enormous capillaries of marrow, we may state their average dimensions as between 7 μ and 10 μ—*i.e.*, when full of blood. The capillaries of the cortex, however, are often not over 4 μ in diameter ($\frac{1}{6250}$ inch), and are therefore of less calibre than the red blood-corpuscle. We must allow for possible shrinking of the vessel by emptying its channel, as well as for the constricting effects of reagents, and can scarcely conclude that even these minute ramifications do not permit the passage of the red blood-corpuscle.

The only constituents of the arterial tunics, which enter into the structure of the capillary, are the **endothelial layer** or **intima** and the **adventitial investment.** In fact, the transition from the smallest artery into the larger capillary is indicated by the disappearance of the muscular fibre cell, and the continuation of the channel as an apparently homogeneous tubular membrane, with oval nuclei along its course, and here and there nucleated connective cells as the sole representative of the adventitial sheath. The intima, which is a direct continuation of the endothelial lining of the arteries and by many believed to be the *only* constituent of the capillary, resembles that lining in every particular save the number and form of its squamous cells. These are not only fewer, being often reduced to two in a transverse view of the vessel or its lumen; but instead of being polygonal, are more often elongated into fusiform plates. These capillaries form good subjects for the study of this endothelial tube after the action of silver nitrate. The darkened cement substance then displays not only the outline of the endothelial plates, but various sized slits and darkened areas termed **stigmata** and **stomata,** and believed by some to indicate orifices through which the colourless corpuscles migrate.

In the smaller capillaries the delicacy of the structure is such that it is at first often overlooked until its course is noticed, mapped out by short, narrow, spindle-shaped nuclei, arranged alternately at regular distances on the opposite sides of the vessel. In the same direction also will be found *rounded* nuclei, staining readily with aniline blue-black, sometimes aggregated into groups or arranged in linear series at very irregular intervals along the vessel. These are the derivatives of the adventitial sheath, and are therefore always external to and placed upon the *fusiform* nuclei. They are often the best guide to the direction of the capillary loops around the nerve cell (Plate xii.).

(3) **The Veins.**—The venous channels of the cortex call but for short notice at our hands, since they reproduce with certain modifications the structures which enter into the formation of the arterial tunics. It will suffice here to show how they differ from the arteries, and to point out the distinctive characteristics of these three divisions of the vascular supply—artery, vein, and capillary.

The veins consist, then, of but three tunics—the Intima, Media, and Adventitia. The **tunica intima** is similar to that of the artery; but the endothelial plates are shorter and broader, and the nuclei rounded and fewer in numbers. The **media** contains no smooth muscle fibre cells, but consists exclusively of connective tissue, whilst the elastic element (always less developed in veins than in arteries) is wholly absent in the small veins of the cortex. The **adventitia** reproduces in all respects what has been already described as constituting this coat in arteries.

Thus we see that the veins may be distinguished from the arteries by the greater *laxity of their tissue*—the absence of the muscular and elastic element leading to a *wider lumen;* moreover, the thin media, due to the absence of muscle cells, results in a *very thin-walled vessel;* in larger vessels the adventitia also is a more prominent feature than the corresponding coat in arteries.

The capillary, on the other hand, commences where the middle coat terminates; but to its minutest ramifications we still find elements of the adventitia around its delicate nucleated wall. This certain authorities deny, but repeated examination leads us fully to endorse this view, also adopted by Eberth, whose views are so much to the point that we quote them here :—

> "Between the capillaries of the hyaloid of the Frog, isolated stellate cells occur, with round nuclei and delicate protoplasm, branching off into many processes, which often anastomose with the processes of the cells of the tunica adventitia. Towards the small arteries and veins, the pericapillary plexus becomes constantly closer, and soon in its stead there appears a delicate transversely folded and nucleated membrane, which is sometimes elevated in the form of small vesicles. . . . A similar nucleated membrane forms the outermost covering of the larger-sized capillaries, and of the arteries and veins of the brain, spinal cord, and retina of man." *

(*c*) **The Neuroglia or Connective Basis.**—The more generally accepted functions of the neuroglia matrix would render structural differentiation of this non-nervous constituent highly probable in

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* "Eberth on the Minute Anatomy of the Capillaries," Stricker's *Histology*, vol. i., p. 286.

Plate V.

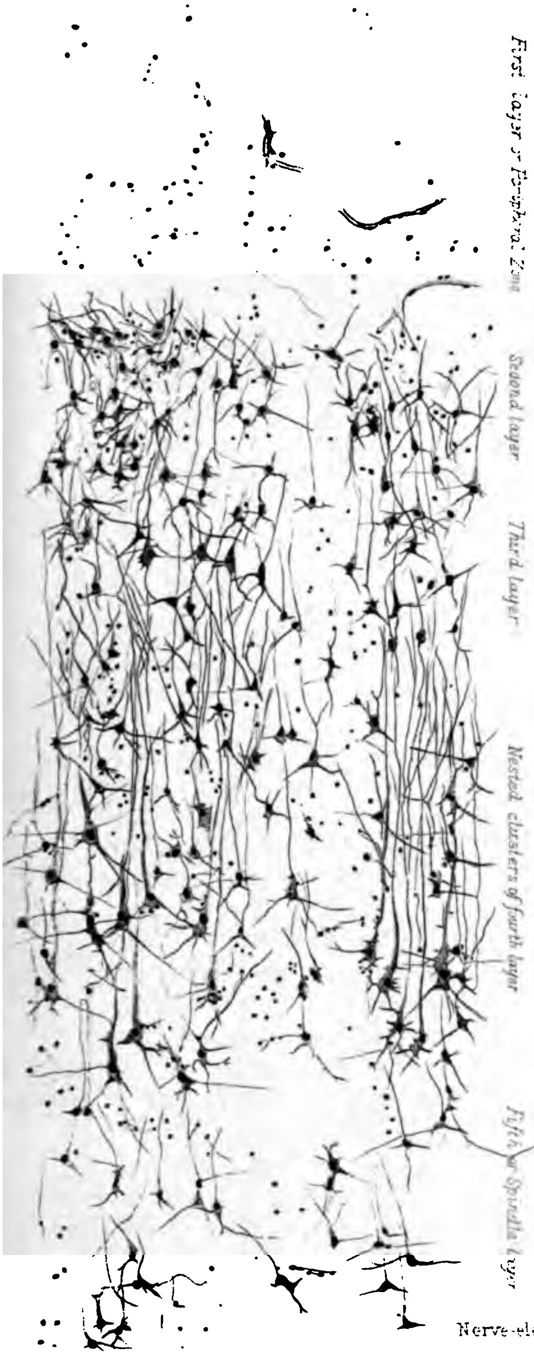
First layer or Peripheral Zone
Second layer
Third layer
Nested clusters of fourth layer
Fifth or Spindle layer

Fig 1.

Motor-Cortex of Pig left hemisphere exhibiting its five laminated type with the Nests of clustered ganglionic cells × 76

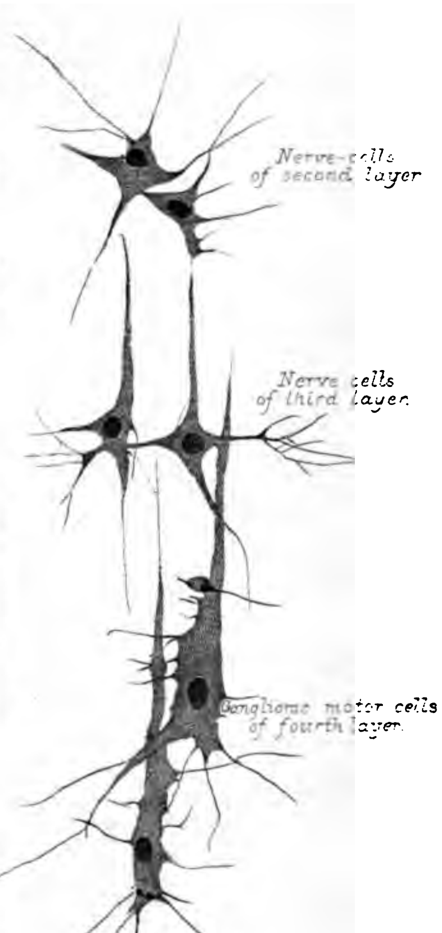
Nerve-cells of second layer
Nerve cells of third layer
Ganglionic motor cells of fourth layer

Fig. 2.

Motor-Cortex of Pig.
Nerve-elements of 2nd, 3rd & 4th layers respectively × 306

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different regions of the cerebro-spinal system. This, we find, accords with actual fact; for, as a supporting, as well as embedding and protective material, the requirements demanded will differ widely in the white medullated structures from those of the grey centres; whilst individual sections of these territories will also differ in the special qualities of this matrix requisite. Thus, the large closely-approximated medullated fibres of the Spinal Cord will be found to possess a strong binding material in the form of large-sized nucleated cells, with numerous lengthened ramifying processes, together with a plexus of fine fibrils (probably elastic fibre – *Gerlach*); whilst a structureless or very finely granular material is found here but sparingly. Still nearer the periphery of the cord, this supporting structure becomes a veritable fibrillar connective sheath of great strength, with trabeculæ of like constitution passing inwards to the cord. In the central grey matter of the cord, however, the finely granular or molecular basis-substance predominates, as most essential for the protection of the extremely delicate nerve fibres present in this region. Farther up in the medulla of the *brain*, as in the neighbourhood bordering upon the grey cortex, the large bundles of medullated fibre again demand a predominance of the connective fibre element, so that here we meet with numerous though delicate ramifying cells. Wherever the medullated fibre reappears, there we find the association of these branching cells, and thus they are seen along the outermost or peripheral layer of the cortex as a normal element. In the grey matter of the cortex, however, the delicate nerve-cell and fibre network appears largely to dispense with this modification of the connective tissue, and we find a structureless matrix vastly preponderating over the cell and nuclear elements of the neuroglia.

A still further modification of the neuroglia element is found on the free surfaces of the cortex immediately beneath the pia, where the branching cell before described fulfils the function of a flattened epithelial investment, whilst the surfaces not exposed to pressure, as the central canal of the cord, show us the element as a columnar epithelium.

Thus, generally, we may affirm that, when dealing with nerve cells and their delicate extensions, the supporting material will be chiefly the structureless or **finely molecular basis-substance**; whilst as we approach the medullated tracts, we shall find that the **connective cell** and **fibre networks** increase at the expense of the former.

The elements of the neuroglia are usually described as nucleated cells and free nuclei imbedded in a structureless, or, according to some, finely fibrillated matrix, and to this view the appearance of chrome-hardened preparations certainly lends support. The less we subject our sections to reagents, and the more recent the section examined, however, the more evident it becomes that the supposed free nuclei are invested by protoplasm, and, in fact, are likewise nucleated cells. These two cell elements differ much as regards their relationships and also their dimensions.

(1) *The smaller of the two kinds of cell* vary from 6 μ to 9 μ in diameter; have a **spheroidal nucleus,** surrounded by an extremely delicate protoplasmic investment, which, as before intimated, is shrunken, often beyond recognition, in hardened specimens. The nucleus is, proportionately to the cell itself, very large, and *invariably stains of an intense depth of colour* with aniline blue-black. These elements appear disposed in three definite situations—(1) irregularly in the neuroglia framework; (2) in regular series around the nerve cells; (3) in more or less regular succession along the course of the blood-vessels (capillary and arteriole).

(2) *The larger cellular elements of the neuroglia* are usually 13 μ in diameter, and supplied with a relatively larger mass of protoplasm as compared with the nucleus. They are distinguished from the former not alone by this greater size and the preponderance of cell over nucleus; but also by their frequent flask-like configuration, as seen *in situ*, and the presence of a *very faintly stained* nucleus, or even sometimes two or three nuclei, observed within them. If these elements are teazed out from the surrounding matrix, they are seen to possess numerous extremely delicate radiating processes; not only the nucleus, but the cell and its extensions are likewise tinted by the aniline dye; not uniformly, however, for the nucleus is always of a slightly deeper tint, but neither cell nucleus nor processes betray anything like the vigour of staining shown by the former element described. The nerve cell, its processes, and the enclosed nucleus had, as we said, a special affinity for this staining reagent, a fact, which indicates very conclusively the *non-nervous* character of these larger elements of the neuroglia. In healthy brain, at least in the human subject, we find these elements chiefly in the outermost layer of the cortex and the central cone of the medulla, but their delicacy, tenuity of branches, very faint staining, and poor differentiation are not favourable to their immediate detection. In certain morbid conditions of the cortex, as we shall see later on, these elements become a most notable and important feature, undergoing excessive proliferation, and betraying their morbid activity by the intensity of colouring which they acquire.

If now we appeal to the silver-chrome methods of preparation, the appearances are very dissimilar. Two forms of cell obtrusively present themselves in both the white and grey matter of the brain; the one with a poorly defined cell body, obscured by much deposit of silver, throwing off on all sides short, shaggy, protoplasmic processes, dendritic or dichotomously dividing; the other, a cell element, also badly defined, forming a centre from which radiate outwards exceedingly fine fibrils, often of great length, sharply defined contour, not dividing dichotomously, nor presenting the thickened hispid aspect of the processes of the first described element. These exceedingly fine fibrils are characterised not only by their length, tenuity, and clean contour, but also by their tendency to exhibit many sharp angular bends along their course.

When the former element is more closely examined, we find the invariable presence of one or more lengthened thick processes by which the cell is attached to a neighbouring blood-vessel; such processes end in a sort of conical or flattened sucker-like thickening on the vessel's walls. We have here, in fact, modified by the silver-chrome method, the same element described as the large cellular element of the neuroglia, and which, later on, we shall refer to as the spider cell. The second form, or stellate cell, has by some been regarded as a distinct element; it is found more particularly in the white matter of the brain, and under the pia covering the cortex. A point of great significance, however, is the fact that they lie invariably in close contiguity to a blood-vessel, and that in favourable preparations, when the dense deposits of silver are dissolving off, we can identify them as spider or Deiter's cells, the vascular attachments being clearly apparent. Between these two forms, superficially so different, there exists a further transitional element readily distinguished in most regions of the brain and medulla, but especially beneath the intima pia, and which at once indicates to us the essential identity of the two former kinds of cell.

The transitional forms alluded to are strung beneath the pia, attached by a vascular process to a vessel of the latter, whilst from the central end of the cell, which is usually ovoid in form, a large number of extremely long delicate fibrils extend into the first and subjacent layers of the cortex, resembling in all respects those of the stellate cell just described. These fibres, however, arise from short stunted protoplasmic processes which are truly dendritic and hispid, and take their origin immediately from the cell body, whilst some of these fine fibrils present numerous moniliform enlargements along their course. To summarise, therefore, we find three stages of development presented by these elements, viz. :—

(*a*) Cells with short, thickened, moss-like protoplasmic processes, dendritic or branching dichotomously, and possessing also thick vascular attachments;

(*b*) Long and exceedingly fine unbranched fibrils radiating from an obscurely marked central cell, also (under certain conditions) showing vascular processes;

(*c*) Transitional forms with vascular processes, short dendritic branches, from which are given off long delicate fibrils like those of the stellate cell, and often distinctly moniliform.

As stated elsewhere, we regard all spider-cells as destined to pass through the three stages—the embryonic moniliform, the fully developed spider-cell or "lymph-connective" stage, and, lastly, the stellate cell; whilst in normal devolution the lymph-connective or spider-cell passes back into the fibre state of the stellate cell, losing its active functional manifestations, and assuming, therefore, the purely mechanical *rôle* of a support for the blood-vessels and medullated nerve fibres in its vicinity.* This study of the constituent histological elements of the cortex prepares us for the consideration of the **lymphatic system** of the brain, and the ultimate relationships of Nerve cell to the Blood and Lymph channels.

(*d*) **Lymphatic System of the Brain.**—To Obersteiner is due the credit of first definitely indicating the existence and relationships of these lymph channels.† Their existence since then has been repeatedly denied, but the evidence hitherto brought forward against Obersteiner's views is most inconclusive in all respects, and in most cases apparently based upon incomplete methods of examination. This is not the place to enter on debateable ground; but we are compelled, owing to the supreme importance of the subject as affecting the physiology and pathology of the brain, to state the results of our own investigations, which were made the subject of a special memoir in 1877.‡

All hardened sections of brain exhibit along the course of their blood-vessels a distinct and more or less wide interval between the vascular walls and the brain-substance; in fact, the brain cortex is channelled throughout, in such a manner, that the vessels when contracted are enclosed within a channel of much greater calibre. The disparity betwixt the diameter of vessel and brain-channel will be affected undoubtedly by corrugating reagents; and hence, we never fail to find these channels disproportionately large in brain which has been subject to extremes of hardening by chromic acid, &c.; but recession of the brain-substance may occur from many other causes acting during life — notably extreme atrophic degeneration; and then, in like manner, such channels will appear inordinately large, however skilfully the brain be prepared. These channels are known by the name of the **perivascular channels** of the brain—the perivascular channels of His: *these are not the lymph channels proper*, as several writers seem to have supposed, but are simple channels in the brain-substance, devoid of an endothelial lining, and communicating freely with the space between the investing pia mater and surface of the cortex, the **epicerebral space.** The adventitial sheath of the blood-vessels becomes closely appressed to this limiting channel, and its (adventitial) nuclei often thus give it the appearance of being lined by endothelial cells. This, however, is not the case, as repeated investigations by silver staining have shown. The student cannot too persistently bear in mind the fact that in these channels he deals purely with what seems equivalent to an involution of the *naked surface* of the brain, and yet the epithelial elements of the epicerebral surface are not continuous along this tubular channel.

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* "Structure of the First or Outermost Layer of the Cerebral Cortex," *Edin. Med. Journ.*, June, 1897.

† "Über einige Lymphraüme im Gehirne" (*Sitzb. d. K. Akad. d. Wissensch.*, Jan. Heft, 1870).

‡ "The Relationships of the Nerve Cells of the Cortex to the Lymphatic System of the Brain," *Proc. Roy. Soc.*, No. 182, 1877.

In the next place we find, under precisely similar conditions to those above enumerated, a wide space around the larger nerve cells; the brain-substance, as it were, seems to have receded from the cell, so that it is enclosed within a circular, oval, or pyriform space. These spaces we will designate the **pericellular sacs.** Genuine sacs, and not mere artificial gaps in the brain-substance, they undoubtedly are, as is abundantly proved by careful examination. To exhibit the true relationships of these perivascular channels and pericellular sacs, let us revert to the smaller cellular element described in the *neuroglia* (p. 94). It was stated that beyond the scattered elements in the basis substance of neuroglia, these cells were arranged in two other directions. Let us particularise:—

(1) The nucleated cells along the arterioles belong to the **adventitial tunic,** and map out its course very accurately; occasionally closely applied to the perivascular channel, as before stated, or separated as irregular ampullæ from the vessel itself, this investment more frequently lies directly upon the **media,** and affords one (but an equivocal) evidence of the existence of a lymph channel surrounding the vessel. That a complete tubular membrane exists for a certain distance along the smaller arterioles is demonstrable; that it is continuous, *as a membrane*, further on to the arterio-capillary plexuses, is more than dubious. It is certain, however, that its representative cells are to be found surrounding these minute channels to their ultimate ramifications; and thus, the perivascular *lymph space* of the adventitia becomes continuous in these districts with the general perivascular channels and sacs around the nerve cells.

(2) The nucleated cells found in connection with the nerve cell in certain states not only accumulate upon the nerve cell itself, but follow closely the outline of the cavity, or, properly speaking, the sac in which the nerve cell lies. Many pericellular sacs will show a

complete series of such nucleated cells around it, still more frequently will they follow out a segment only of its circular outline; occasionally none may be seen—an exception due probably to displacement during section-cutting or further manipulation. Upon closer observation, however, it becomes apparent that in the immediate neighbourhood of every large nerve cell there is a minute arteriole or capillary, not indicated so often by a well-differentiated contour (for these minute vessels are usually most difficult to follow) as by the direction of its nucleated cells. Thus, the fusiform nuclei of the intima, alternately placed on opposite sides of the capillary, will lead to the discovery of the outline of the vessel faintly indicated in a graceful curve or spiral in close approximation to the nerve cell; but the presence of the deep-stained nuclei of the adventitial cells taking the same course, plainly indicates the direction of these ultimate nutrient channels. It is these adventitial elements which give us the clue to tracing the obscurely marked capillary, and when this is followed out, the eye becomes accustomed to trace without any difficulty the *vascular loop* around the nerve cell.

Around a segment of the pericellular sac, mapped out by adventitial elements, we then see a delicate tubular loop, evidently continuous with the neighbouring arteriole, and to the sides of which the pericellular sac appears to be attached, the nerve cell itself being, as it were, suspended within the latter. It would appear as if the general perivascular channels at their ultimate ramifications around the arterio-capillary plexuses were enlarged here and there laterally along the vessel by the growth of an element included within it which becomes the nerve cell, and which does not come in contact with the neuroglia matrix except through the medium of its processes, which, passing through the pericellular sac, permeate the neuroglia in every direction. It would appear also from examination of specially prepared sections, that the adventitial elements are not entirely limited to the vascular loop, but may line the interior of these sacs—not as a regularly applied endothelial layer, but as loosely distributed and branching cells. In like manner, similar cells may be found free within the cavity of the sac between its wall and the nerve cell, resembling in all particulars lymph corpuscles.

Beyond the system of perivascular channels, adventitial lymph space, and pericellular sac, we have a **lymph-connective system** which plays an important *rôle* in the pathology of the brain. This system is constituted by the larger connective element referred to above—the delicate branching masses of protoplasm supplied usually with one, sometimes with two, or even three large nuclei. These elements, when more closely examined, are found, as already indicated, to have a definite and constant relationship to the cortical blood-vessels; and are always discovered in larger numbers in their immediate neighbourhood, external to the perivascular channels. The latter present, where they are well seen and the adventitial sheath is appressed to the vessel's side, a series of delicate processes, which, traversing the channel, look like fibres extending from the adventitia into the brain-substance.

What are these fibrous prolongations? Careful examination of one of the large neuroglia elements reveals the fact that they throw off *two sets of processes*—(1) an enormous number of *extremely delicate* fibres, which spread into the intervascular area around, and (2) a *much thicker, coarser process,* which, often after a tortuous course, *ends in the adventitial sheath* of the blood-vessel. In crossing the perivascular sac, these processes give rise to the fibres just described as extending between adventitia and brain-substance.

It is in certain morbid developments of these cells that we can the more readily distinguish their real relationships. We find that the stouter process, which we may provisionally term the **vascular,** terminates in a nucleated mass of protoplasm on the sheath itself. In morbid states, as we shall see, this terminal protoplasm of the vascular process becomes spider-like, in its turn throwing off numerous branches, which embrace the vessel's wall. In the healthy state, it is most difficult to trace the vascular branch; but that this *can* be done by proper methods, we have frequently satisfied ourselves. The branched cells which we have now described have often been recognised in their morbid modifications, and variously interpreted. Their representatives in healthy brain were first described by Deiters,[*] and subsequently by Ball and Golgi; but we do not think their true significance has been recognised either as normal or pathological elements of the central nervous system. We incline to regard these elements as comprising the **distal extension of a lymphatic system,** in fact as a **lymph-connective system** permeating the neuroglia in the intervascular area. The individual elements are excessively delicate and pellucid, their protoplasm appearing almost of fluid consistence, and the vascular process invariably establishing its connection with the lymph sheath of a blood-vessel. In whatever manner these spider cells effect the reabsorption and distribution of the effete material and surplus plasma—whether by direct assimilation into their own structure, and its removal by currents within the protoplasm of the cell and its processes, or by means of a true canalicular system terminating in the lymph sheath—it is an undoubted fact that any arrest to the escape of perivascular lymph from the cortex is immediately followed by a morbid development and hypertrophic condition of this system of **spider cells,** as we shall for the future call these elements of the "lymph-connective system."* Meynert long since drew attention to their frequent presence as associated with congestion and degeneration of the lymphatic glands of the head and neck, and we have assured ourselves of the frequent association of this morbid development in tuberculosis, and in several affections of the cortex and its membranes which lead to obstruction of the perivascular lymph channels.† The morbid changes undergone by this lymph-connective system and the effects of its morbid activity will be more fully dealt with when treating of the pathology of the cortex.‡ For the present we shall summarise the above statements as follows :—The lymphatic system of the brain consists—

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[*] Hence they are often named after him—Deiters' cells.—*Untersuchungen über Gehirn und Rückenmark der Menschen und der Säugethiere,* 1865.

(1) In the first place, of a distensible lymphatic sheath, loosely applied around the arterioles and venules, containing numerous nucleated cells in its texture—the *adventitial lymph sheath*, the whole being included within a non-distensible channel of the brain-substance, devoid of endothelial lining—*perivascular channel of His.*

(2) In the second place, of a continuation of the cellular elements of this sheath, loosely applied to the **arterio-capillary plexuses,** still contained within a perivascular channel, which now exhibit along the capillary loop sac-like dilatations—the *pericellular sacs*, within which the nerve cell lies, surrounded by plasma.

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* It may prove of interest to give here references to some few of the articles bearing directly upon phagocytosis :—"Poisonous and Defensive Albumoses," by Dr. Hankin in *Brit. Med. Journ.*, May, 1890, p. 126. "Lectures on Phagocytosis and Immunity," by Sims Woodhead, *Lancet*, Jan. and Feb., 1892. "Discussion on Phagocytosis and Immunity," *Brit. Med. Journ.*, Feb. and Mar., 1892. "Ptomaines and Animal Alkaloids," *Brit. Med. Journ.*, Nov., 1892. "The Spider (so-called Scavenger) Cell of the Brain," by Edwin Goodall in *Journal of Pathology*, Feb., 1894. "The Spider or Phagocyte Cells of Senile Insanity, General Paralysis, and Alcoholic Insanity," by Alfred Campbell, *Journ. Mental Sc.*, Oct., 1894.

† We have elsewhere alluded to the comparative significance of these elements as follows :—"In man they appear in scanty numbers ; in the Barbary ape, they become more frequent ; in the cat and ocelot, they are still more abundant ; in the pig and sheep so profusely scattered are they that they form a most characteristic stratum immediately below the pia mater, and the meshwork formed by their fibres is dense and coarse, binding the blood-vessels to the cortex and rendering the pia mater strongly adherent. We find these corpuscles in human brain which has undergone senile degeneration—in other diseases attended by reduction in functional activity, and in vascular affections resulting in retrogressive changes and a reversion to a low type of structure."—"Comparative Structure of the Cortex Cerebri." *Trans. Royal Soc.*, part i., 1880.

‡ See in this connection a suggestive article by Dr. Shaw on Aprosexia in Children—a condition where adenoid growths (post pharyngeal) obstruct the flow of lymph from the frontal lobe, which naturally escapes through the lymphatics of the ethmoid plate.—*Practitioner*, July, 1890.

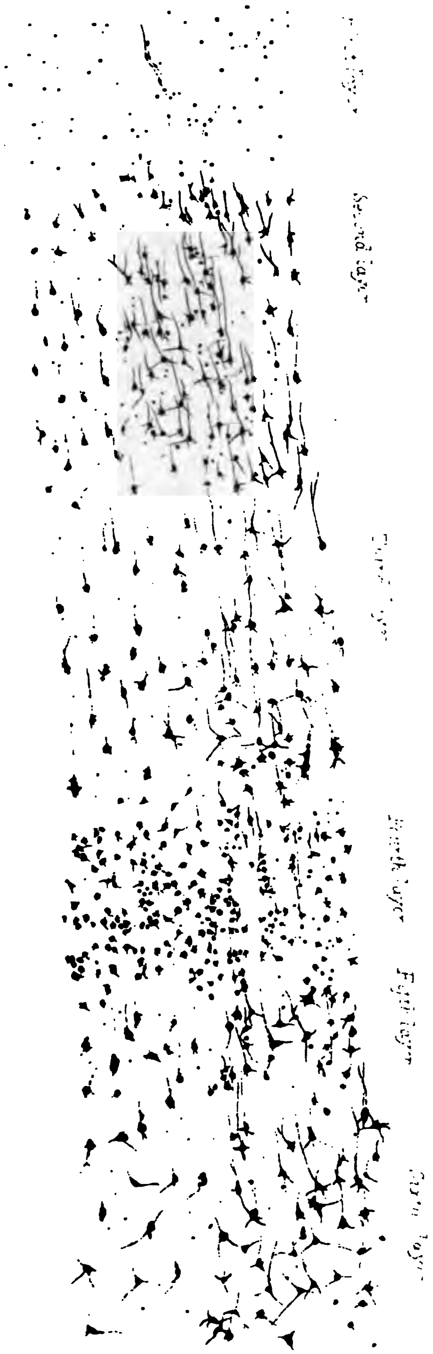

Fig. 1.

'Sensory type"
Section through First Annectant
Gyrus of Human Cortex. ×65.

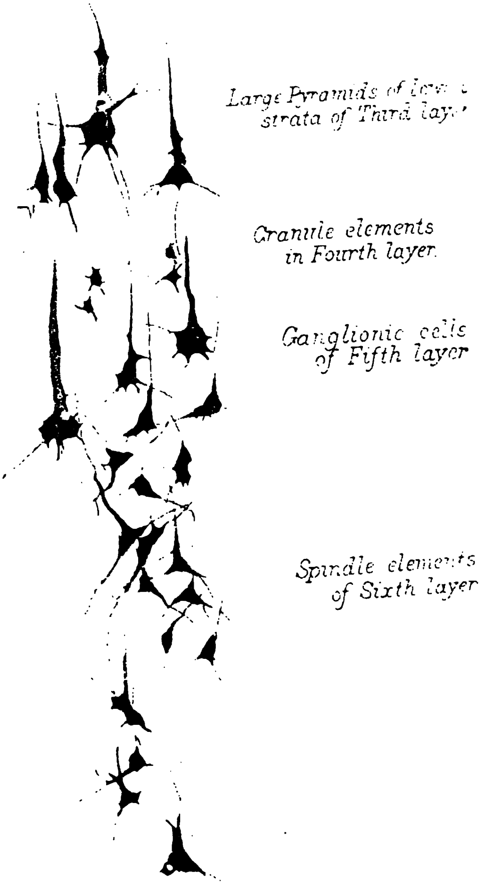
Large Pyramids of lower strata of Third layer
Granule elements in Fourth layer
Ganglionic cells of Fifth layer
Spindle elements of Sixth layer

Fig. 2.

"Sensory type"
Nerve elements of 3rd 4th, 5th & 6th layers
of First Annectant Gyrus of
Human Brain.
×157.

Bale & Danielsson Ltd Sculp

(3) Lastly, of a system of plasmatic cells with numerous prolongations, which are always in intimate connection with the adventitial lymph sheath, and which drain the areas between the vascular branches: these we have termed the *lymph-connective elements.*

If we take a comprehensive view of the whole system—the channelled vascular tracts, the saccular ampullæ along the capillary tube, the canalicular-like formation of the lymph-connective elements, all embedded in a homogeneous matrix of neuroglia—we cannot but be struck by the *sponge-like* arrangement of the cortex, and the facilities so afforded for the free circulation of plasma throughout its most intimate regions.

CORTICAL LAMINATION.

Having familiarised himself with the individual histological elements of the cortex—the nerve cells, blood-vascular and lymph-vascular systems, and the neuroglia framework—it becomes the student's duty to examine their general arrangements and the local deviations to be observed.

A vertical section of fresh cortex of human brain reveals to the naked eye a distinctly laminated aspect, the various laminæ of which are more or less clearly marked out by difference in colour, the outer being usually of a pale translucent grey, and the deeper of alternating pale and dark grey layers, more opaque in aspect, and in certain regions exhibiting a sharply-defined white streak. The outer translucent layer has superimposed on it a delicate white stratum, scarcely appreciable on the convexity of the hemisphere, but well-marked in the convolutions bordering upon the corpus callosum, and the convolution of the hippocampus, at the base, where its peculiar aspect has gained for it the name of the **reticulated white substance.** As we shall see later on, this is a superficial layer of white medullated fibre running *parallel to the surface* of the convolution; whilst the paler intersecting streaks deeper down in the cortex are similar systems of arciform intracortical fibres intervening between layers of grey substance. The deeper layers owe their opacity to the relatively large proportion of medullated fibres passing through them; the upper layers are translucent from the preponderance of the neuroglia element and fine protoplasmic processes of the nerve cells; the warmer grey tints are due not only to large numbers of pigmented nerve cells, but chiefly to the amount of blood in the vessels of the layer.

As might be supposed from the above, the distinctness of lamination not only varies with the local peculiarities of structure, but with morbid states of the cortex and with the full or empty state of its vessels. Probably the best introduction the student can have to the study of the human cortex is to commence first with the brain of one of the lower mammals, choosing one of the smooth non-convoluted brains, as of the rat or rabbit, ere he attempts the more complicated brain of those animals which exhibit a convoluted surface. He thereby learns to appreciate the great diversity of lamination which may exist in so small an organ as the brain of the rodent, as also the abrupt transition from one type of cortex to that of another wholly different from it, and lastly he becomes familiar with types of lamination which are strictly reproduced in higher forms up to the brain of man.

Figs. 1–3 in Plate i. represent the brain of the rabbit seen from its upper, lower, and median aspect, of somewhat pyriform contour below at the base, and triangular above; its frontal pole is much attenuated, and rests upon the olfactory lobe. On its inner aspect we see two very delicate furrows (fig. 1, A) which represent the sub-frontal and sub-parietal segments of the limbic fissure, which is strongly marked in the brains of the pig and of the sheep; this rudimentary fissure limits the **upper limbic arc** (between A and J) from the **extra-limbic** or **parietal mass** of the hemisphere (fig. 3, Z, Y).

If we follow this upper limbic arc from before backwards, we find that its anterior extremity is deep, and that it gradually becomes more shallow towards the sub-parietal furrow; beyond this it is hollowed out by the prominence of the mesencephalon and overhung by the occipital pole (fig. 1, D), and curving downwards behind the corpus callosum, it bends forward as the gyrus hippocampi or lower limbic arc (figs. 1, 2, B).

Looked at from the base, we see the lower limbic arc separated from the extra-limbic mass by a well-defined fissure—the limbic fissure, which here separates the lower limbic arc from the extra-limbic mass, the latter being still prominent and *not* concealed from this aspect, as in the rat, where the lower limbic arc extends farther outwards. Extending back from the frontal pole are the olfactory lobes, the outer roots of which (or superficial olfactory medulla) terminate near the extremity of the gyrus hippocampi. These two external olfactory roots enclose between them two pyriform grey areas, one on each side, separated by the middle line, bounded behind by the optic commissure—the optic nerves lying superficial to them. This grey area is the olfactory field of Gratiolet. Between the olfactory area and the lower limbic arc, a very slight depression indicates the site of a rudimentary Sylvian fissure.

Looked at from above, we find the surface of what Broca would call the extra-limbic portion, perfectly smooth, and showing no indications of rudimentary furrowing beyond a very delicate, shallow, linear depression, mapping off the sagittal region of the brain from the parietal or extra-limbic portion in the posterior half of the hemispheres. This is the representative of the primary parietal sulcus, which in the Pig, Sheep, and other Gyrencephala, separates the sagittal from the sylvian gyri of the parietal lobe. In the rat no such linear depression exists; but, this region bordering on the sagittal margin posteriorly, *is clearly mapped out by its distinctly pale aspect* as compared with the cortex external to it.

The different regions which we have now indicated are all distinguished by a type of cortex peculiar to each; and thus the upper limbic arc, the lower limbic arc, the olfactory area, the extra-limbic or parietal portion—areas obviously differentiated roughly from one another by sulci or faint indications of furrowing—all exhibit absolutely distinct types of cortex. But this differentiation does not stop here; the pale strip of cortex bordering upon the sagittal margin in the rat, although not mapped off by a distinct furrow, has also its own peculiar type of cortex; and in the rabbit, as we have seen, this region is further differentiated by a linear furrowing. Then, again, the lower limbic arc, if traced backwards, presents us beneath the occipital pole with a further modification, which can only be regarded as a distinct type of cortex. If we add to the above the formation of the cornu Ammonis and of the olfactory bulb, we have presented to us *eight* distinct types of cortex, not mere fanciful distinctions based upon trivial peculiarities; but, in all cases, *abrupt transitions* from one *kind* of cortex to another. This divergence in *laminar type* is peculiarly abrupt in these lower forms of life, the demarcation usually being sharply drawn at the furrows intervening between these regions. In higher animals, and especially in man, no such abrupt demarcation occurs; distinct **transition regions** lie between either territory, so that the *gradual passage from one form of cortex to another* is a distinctive element in the evolution of the higher brains* (*Brain*, vol. i., page 84). The eight laminar types of cortex which are thus distinguishable in these small mammalian brains, we have named as follows:—

(1) Type of the upper limbic arc.
(2) Modified upper limbic type.
(3) Outer olfactory type.
(4) Inner olfactory type.
(5) Modified olfactory type.
(6) Extra-limbic type.
(7) Type of cornu Ammonis.
(8) Type of olfactory bulb.

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* In his earlier memoir, published in Stricker's *Human and Comparative Histology*, as well as in his later views expressed in *Psychiatry*, Meynert defines but five types of cortical lamination as distinctive of the brain in mammals. We find ourselves unable to agree with Meynert, not only as regards his enumeration of types of lamination, but in some cases as regards his description of the specific characters of individual types of cortex.

On the other hand, we find that Meynert enumerates but five types as follows:—

(1) Common type. (3) Sylvian type.
(2) Occipital type. (4) Type of cornu Ammonis.
(5) Type of olfactory bulb.

In addition to these types of cortical lamination we have also to consider the cerebellar cortex, and what is really an offshoot of the cerebral cortex—viz., the retina.

If we turn to our outline scheme of the rabbit's brain (Plate i.), we shall find these diverse forms of cortex distributed in the following regions:—

(1) The first, or the type of the **upper limbic arc**, occupies the median cortex of the hemisphere from the frontal pole to the end of the sub-parietal furrow (figs. 1–3, +); it moreover spreads beyond the sagittal margin, and embraces the pointed frontal extremity of the extra-limbic region at the vertex.

(2) The second, or **modified upper limbic type**, prevails also on the median cortex behind the above type, extending to the occipital pole, but also spreading outwards over the sagittal border to the upper aspect of the hemisphere, where it terminates abruptly at the parietal furrow (dotted area).

(3) The third, or **outer olfactory type**, characterises the cortex of the greater segment of the lower limbic arc to its extremity—the gyrus hippocampi (figs. 1, 2, B).

(4) The fourth, or **inner olfactory type**, covers the grey pyriform areas enclosed within the last mentioned and the outer root of the olfactory bulb (fig. 2, dark area).

(5) The fifth, or **modified olfactory type**, occupies the posterior segment of the lower limbic arc, where it sweeps round posteriorly to meet the upper limbic arc. This form of cortex, unique of its kind, is also abruptly limited externally by the great limbic fissure.

(6) The sixth, or **extra-limbic type**, is peculiar to the whole of the extra-limbic or parietal portion of the hemisphere, except the regions already described as presenting a peculiar lamination. Thus it occupies the whole of the vertex except the portion internal to the parietal furrow, and the pointed end of the hemisphere in front, whilst elsewhere it is strictly demarcated from other regions by the great limbic fissure.

(7) The seventh, **or type of the cornu Ammonis**, characterising the involuted free margin of the cortical envelope, is, of course, concealed from view in these aspects of the hemisphere.

(8) The eighth, or **type of the olfactory bulb**, has its distribution sufficiently indicated by its name (figs. 1, 2, F).

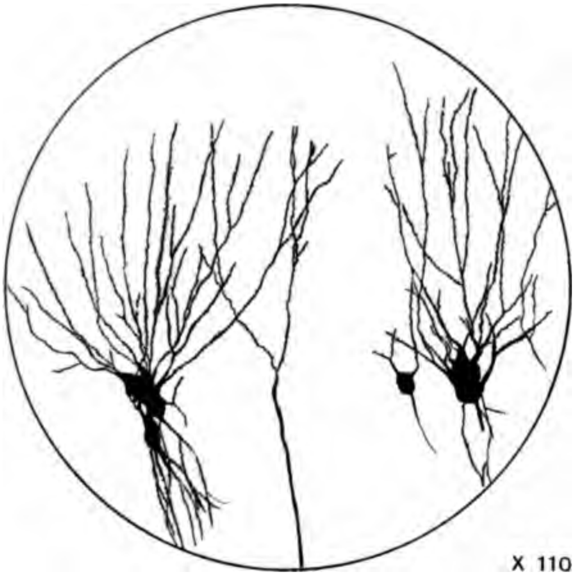
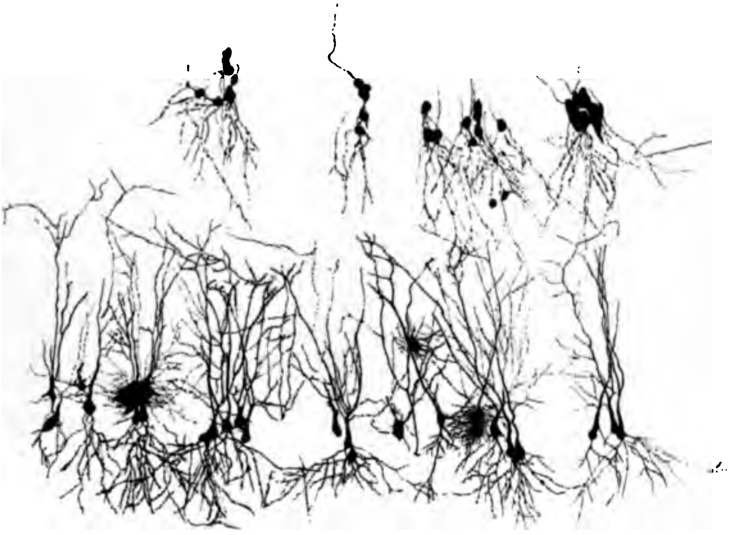


FIG. 1.



X 110

FIG. 2.

CORNU AMMONIS OF YOUNG RAT. SHOWING PYRAMIDAL CELLS OF CORNU, AND GRANULES OF FASCIA DENTATA.

Bale, Sons & Danielsson, Lᵗᵈ

A brief description of the peculiarities of these cortical belts of nerve cells will be all that is needful for our present purpose.

(1) **Upper Limbic Type.**—The cortical lamination here referred to is illustrated in Plate i. The area it covers is represented in figs. 1-3, +.

It is essentially a four-laminated type; its first or superficial layer being a light grey belt of delicate neuroglia matrix, with connective elements and their fine prolongations supporting the extremely delicate subdivisions of the apical processes of nerve cells in the subjacent layers. This layer we term the "peripheral cortical zone" (Plate i.). Next to this succeeds a layer of small pyramidal cells, which, down to the confines of the third layer, remain equable in size throughout; in all respects these elements bear close resemblance to the upper half of the third layer in higher animals. They differ from the human cortex (1) in not, as in the latter, rapidly increasing in size with their depth, and (2) in following immediately upon the peripheral cortical zone with no intervening belt of small oval and angular cells, such as characterises the second layer in man. A few bifurcate cells in sparse detached clumps occur on the outermost confines of this layer, probably rudimentary elements of the second layer of man. Beyond the layer of small pyramidal cells is a pale belt containing the largest cells of the cortex—a pale poorly-celled zone demarcating them from the superimposed layer of pyramidal cells. These elements are, however, distinguished from the latter not alone by their great size, but by their distribution into confluent groups or clusters, which, as we shall see later on, is a special character of the large nerve cells of the motor cortex. Their apex process extends right through the pyramidal series into the peripheral zone. We cannot now stop to inquire into their many striking features. Beneath these large cells is a series of fusiform elements similar in all respects to those found in higher mammals. This type of cortex, therefore, is constituted by

(1) A peripheral cortical zone.
(2) Small pyramidal layer.
(3) Ganglionic layer.
(4) Spindle cell layer.

(2) **Modified Upper Limbic Type.**—This form of cortex, like the last is also a four-laminated type. Near the posterior extremity of the corpus callosum (Plate x., fig. 1), the upper limbic arc exhibits the **intercalation** of a series of **granule cells** *between* the small pyramidal and the large ganglionic cells; but, as we proceed farther back, this belt of granule cells deepens, and, approaching the surface, eventually entirely displaces the small pyramids, and becomes in their place the second layer in this region. The granule-like aspect is due to the relatively large nucleus, as compared with the investing protoplasm: they form a belt of densely crowded elements. The cortex, therefore,

of the area represented in Plate x. (figs. 3 and 5, dotted area) is constituted of

(1) Peripheral cortical zone.
(2) Deep belt of granule-like cells.
(3) Ganglionic belt.
(4) Spindle cell layer.

(3) **Outer Olfactory Type.**—Passing now to the lower limbic arc at the base, we find that the area marked Plate i., B, has a much simpler form of cortex than those hitherto described—two belts of nerve cells only are found in this region subjacent to its outer or peripheral zone. This peripheral zone is specially characterised by the distribution throughout its greater extent of fibres derived from the superficial olfactory fasciculus, which lies embedded in this first layer of its cortex; fibres which ramify at all depths in this layer to unite with the meshwork derived from the apex processes of the cells beneath. Next to this succeeds a shallow belt of irregular cells, pyramidal, oval, or fusiform, small in size, each with a bifurcate apex process, which immediately undergo rapid subdivision. They are arranged in peculiarly appressed clumps. Then amongst them appear a few large cells of pyramidal contour, which deeper down increase in number and form a distinct belt, in which a few rather large elements are seen. Traced outwards, beyond the limits of the great limbic fissure, these larger elements appear to pass into the ganglionic series, whilst the small clumps of irregular cells pass into the small pyramidal cells of the extra limbic region. This cortex, therefore, comprises

(1) A peripheral cortical zone.
(2) Dense appressed clusters of small cells.
(3) Scanty large pyramidal cells.

(4) **Inner Olfactory Type.**—Covering Gratiolet's "olfactory area" is a three-laminated cortex, comprising

(1) A peripheral zone.
(2) A granule cell layer.
(3) Layer of spindle cells.

The second layer is formed of cells measuring $9\mu \times 6\mu$, with a large spheroidal nucleus, 6μ in diameter; with these are associated numerous minute granules only 5μ in diameter, like the granule cells of the modified upper limbic region. This layer is duplicated in numerous folds, in which the outer layer does not participate. The layer of spindle cells is notable for the large size of these elements; they are reclinate—*i.e.*, their long axis lies parallel with the surface of the cortex.

(5) **Modified Lower Limbic Type.**—This unique formation, occupying the small triangular area, shown in the figure (Plate i., T), is a five-laminated type, the chief feature of which is presented by the peculiar second layer of cells. These nervous elements are more than double

the size of those occurring in the second layer of the cortex elsewhere; they are large, swollen, globose, inflated-looking cells, which almost invariably branch from the apex by a bifid or bicorned process. This belt of inflated cells is superimposed on a series of *small* pyramidal bodies, which succeeds them (Plate x., fig. 2). A pale belt, devoid of nerve cells, follows the latter, and is in turn succeeded by a series of spindle cells. To recapitulate, we have here

(1) Peripheral cortical zone.
(2) Layer of globose inflated cells.
(3) Small pyramidal cells.
(4) Pale belt devoid of nerve cells.
(5) Spindle cell layer.

(6) **Extra-Limbic Type** differs from that of the upper limbic cortex solely in the intercalation of a belt of granule or angular cells between the small pyramidal and ganglionic series. This form of cortex exhibits a very gradual transition to the upper limbic type, and, therefore, presents an exception to the rule of abrupt demarcation shown by other varieties of cortex. The gradual passage of one into another form we shall have reason to refer to later on; for the present, it will suffice to enumerate the relative layers of this formation.

(1) Peripheral cortical zone.
(2) Small pyramidal layer.
(3) Belt of granule or angular cells.
(4) Ganglionic series.
(5) Spindle cell series.

(7) **Type of the Cornu Ammonis.**—The cortex of the cornu presents several features common to other regions of the hemispheres: we here have reproduced a peripheral zone to which run the radiate apex processes of underlying cells: then a dense belt of ganglionic cells: beneath which again we trace a spindle-form series of elements. The distribution, however, of these several nervous constituents is so far different as to stamp this type of cortex with features peculiarly its own.

Peripheral Zone (*Limiting Zone or Molecular Layer*).—This superficial layer receives the terminal dendrites ascending from the subjacent pyramidal cells of the fourth layer, and the terminal ramifications of axons also derived from subjacent cells—**ascending axons.** Apart from these, however, we find, according to Cajal, certain nerve cells *proper* to this layer—cells of fusiform or more or less triangular form, with several dendritic branchings, whilst a fine axon ramifies very extensively through the whole thickness of the stratum. Meynert recognised spindle-formed cells in this his "Nuclear layer."

The cells whose axons enter into the constitution of this peripheral zone are the nerve cells horizontally disposed within the second layer—the so-called "lacunar layer" of the cornu; as well as the polymorphic cells of the fifth layer, lying just beneath the belt of pyramidal cells.

Lacunar Layer.—This, the second layer of the cornu Ammonis, is formed by

(1) Horizontally disposed medullated fibres, the origin of which has been traced to the large size collaterals ascending from the axons of the giant pyramids and ramifying across the primary dendrites of the small pyramids, which run vertically at this level;

(2) Terminal arborisations from collaterals ascending from the white matter or alveus;

(3) Terminal arborisations of the ascending axons of subjacent cells;

(4) The intrinsic cells of this layer, usually triangular in form, possessed of several dendrites, and an axon which terminates in an arborisation horizontally distributed along this layer, losing itself, as before stated, in the lower region of the peripheral zone.

Striate Layer.—This is the part characterised by its radiate appearance due to the ascent of the dendrons of the pyramidal cells. Several species of nerve cell are found in this layer, distinguished by the distribution of their axons. All have dendritic expansions, which are distributed to the two upper layers on the one hand, and to the layer of polymorphic cells on the other. All such cells give origin to axons which ramify extensively; some upwards into the two superficial layers; others horizontally within the striate layer; and, lastly, others descending to end in free arborisations around the pyramidal cells and the subjacent polymorphic cells (*Cajal*).

Pyramidal Layer.—These nerve cells, so characteristic of the cortex of the cornu Ammonis, vary much in form in different animals; they are closely appressed and often appear in several tiers one over the other. More frequently oval or spindle-shaped, they, at times, assume a more spherical contour; but, in all cases, whilst several protoplasmic processes descend from their base, an apical dendron ascends towards the peripheral zone. This latter throws off several collaterals on its ascent through the striate layer, and upon its arrival at the lacunar layer, its primary splitting up into dendrites occurs, and a rich terminal plume results, whose branches extend up to the pial surface of the cortex. Like the corresponding pyramids of the cerebral cortex these dendrites are covered by rough thorny projections. The axon of these pyramids (long known to be continuous with the medullated fibres of the alveus) descends to the white stratum below, throwing off several collaterals in their course, and then, bending at right angles, becomes continuous with the medulla of the alveus—many bifurcating into two branches which run in opposite directions.

A very important distinction must be made between the inferior or giant pyramids near the fimbria, and the superior or small-sized pyramids beneath the alveus as regards their conformation, relationships, and functional significance. The former are not only larger in size,

Plate VIII.

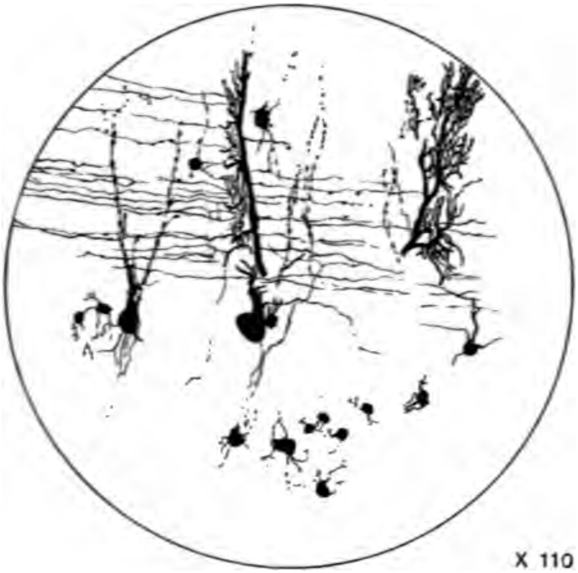


FIG. 1.

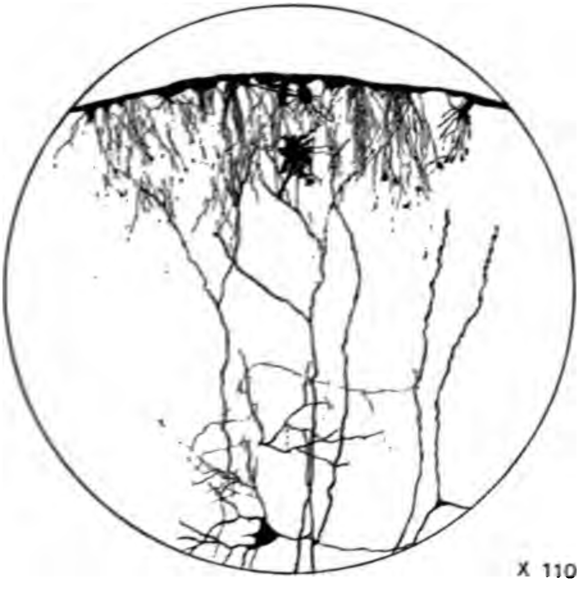


FIG. 2.

Bale, Sons & Danielsson, Ltd., Lith.

have thicker dendrons with stunted dendritic arborisations, but also their axons are continuous with the fibres of the fimbria, not with those of the alveus as is the case with the smaller pyramids; at the same time these axons *originate the recurrent collaterals* already alluded to, which pass through the lacunar layer to ramify over the primary dendrites of the smaller pyramids. A further distinction is established by the fact that the axons of the granules of the fascia dentata (moss fibres) are brought into close relationships with the dendrons of these giant cells alone and not with those of the smaller cells.

Polymorphic Cells.—The irregular and fusiform cells *beneath* the pyramidal layer constitute the "polymorphic layer" of Cajal—the old "stratum moleculare" of Kupffer. Formerly classed as simple spindle-shaped cells recent authorities describe certain notable features as presented by them. They may be divided into three groups—all the cells of which are dendritic—these dendrites ramifying amongst the protoplasmic processes and the collaterals from the axons of the pyramids. These groups are characterised as having—

(1) Ascending and recurrent axons;
(2) Horizontal and plexiform axons;
(3) Deep spindle cells with ramifying axons.

In the first group of ascending and recurrent axons this process ascends through the radiate stratum, throwing off occasional collaterals in this course, some of which pass up into the lacunar and peripheral layers, whilst the main axon curves downwards again and, passing into the pyramidal layer, forms a rich terminal plexus of fibres around these cells. In the second group, the axon passes off horizontally and divides into numerous branches after the manner of Golgi's sensory cells. From these originate collaterals, which ascend to the pyramidal layer and surround its cells with a plexus of branches mingling with the similar plexus from the recurrent axons.

In the third group, the cells are spindle shaped; they have long been recognised in the neighbourhood of the alveus, but only recent research has revealed the fact that they possess axons which ramify extensively in this stratum and ascend upwards to a higher level.

Besides these three, Cajal describes a cell which gives off an ascending axon terminating by ramification in the lacunar and peripheral layers, but having no recurrent branch. We need scarcely include here certain cells, as Cajal does, which, together with others in the stratum radiatum, are admittedly similar in type to the pyramidal cell, and are really *dislocated* cells not special to these layers, any more than we should speak of those cells found in the lowermost stratum of the peripheral layer which are really elements dislocated from the cells of the second layer of the cerebral cortex, and which are also seen in the region of the fascia dentata.

Medullated Layer or Alveus.—This deepest layer, bounded centrally by the ependyma of the ventricles, is largely constituted by the axons of the pyramidal cells, many of which, as before stated, bifurcate into a thick and a slender process running in an opposite direction. These medullated fibres throw upwards collaterals which ramify in the upper layers of the cornu.

The Fascia Dentata.—In this region we have to distinguish three layers, viz. :—

(1) Peripheral or molecular;
(2) Granule layer or small pyramids;
(3) Polymorphic cells.

Molecular Layer.—This, like the corresponding layer of the cortex generally, and that of the cornu Ammonis formation proper, receives the terminal plume of dendrites given off from the subjacent cells, and which are here brought into relationships with the axons of certain nerve cells peculiar to this layer. These cells are distributed in a double series, superficial and deep (*Cajal*); they are ovoid, stellate, or spindle-shaped—the deeper cells being the larger and more freely supplied with protoplasmic processes. Both have an axon thicker in the deeper cells, and ramifying to great distances in a horizontal direction, but much finer in the smaller cells and far more restricted in range.

Stratum Granulosum.—The elements of this layer are very small, densely appressed, forming a deep stratum of minute ovoid, pyramidal cells, throwing off numerous protoplasmic processes towards the peripheral zone: whilst its axon, descending through the subjacent layer of cells (polymorphous), gives origin here to numerous delicate collaterals, which ramify amongst the polymorphic cells. Continuing its course, it bends as a knotty, non-medullated fibre along the length of the cornu, upon the body and dendrons of the giant pyramids. These axons from the granules appear similar in all respects to the so-called "moss fibres" of the cerebellum. Cajal affirms that such moss fibres never trespass on the region of the alveus or the lacunar layer of the cornu proper; but are invariably confined to the giant pyramids, their moss-like bunches resting in close contact with these cells. It will be observed that these axons of the granules form a terminal arborisation, bringing them into relationship with the giant pyramids and their dendrons, just as the collaterals ascending from the axons of the latter ramify over the dendrites of the smaller pyramids of the cornu.

Pyramidal cells of notable character occupy the upper regions of this granule zone. They have an apical process (protoplasmic), which ascends to be distributed in branches to the molecular layer; several protoplasmic branches given off from the base of the cell, and an axon which running horizontally immediately above the granule layer sends downwards numerous collaterals to envelope the granules in a rich plexus of branches.

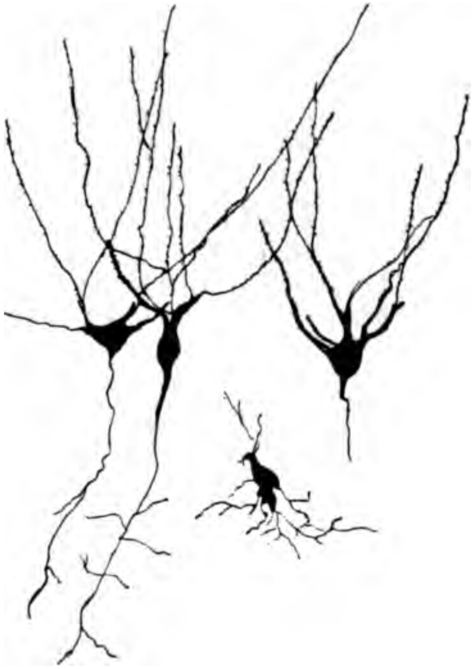


Fig. 14.—Cerebral cortex : granules of fascia dentata—Cornu Ammonis (rabbit).

Stratum of Polymorphic Cells.—Irregularly shaped cells are seen beneath the granule layer, throwing off protoplasmic processes in several directions, and with an ascending axon passing upwards through the granule into the molecular layer, there to bifurcate and ramify in extended horizontal planes. Some of these cells are stated, however, to give off an axon which runs horizontally along the summit of the granule zone, taking part with the pyramidal cells in the formation of the intergranular plexus already described. Cajal, moreover, describes in this layer nerve cells similar to the sensitive cells of Golgi; and others with a *descending* axon continued into the alveus. Lastly, immediately above the molecular layer of the cornu proper, where it is concealed by the fascia dentata, are large fusiform cells, whose axon can be followed into the alveus.

The Olfactory Bulb.—The cortex of the olfactory bulb consists of a superficial and a deep fibre-tract; the former non-medullated, connecting the nervous apparatus of the Schneiderian membrane with the olfactory bulb; the latter, medullated, and connecting the bulb through the olfactory tract with the centric termini in the cerebral cortex. These two tracts of nerve fibre are brought into relationship through the medium of the intervening layers, three in number, of nerve cells and fibre systems.

We have thus, from without inwards, five distinct strata:—

(1) Flexus of peripheral nerve fibres.
(2) Layer of olfactory glomeruli.
(3) Molecular layer with small nerve cells.
(4) Layer of so-called "mitre cells."
(5) Layer of granules and medullated nerve fibres.

The superficial layer of the bulb is formed by non-medullated fibres which, arising from bipolar nerve cells of the Schneiderian membrane, pass through the cribriform plate to form a densely woven plexus, on the surface of the bulb; the fibres eventually terminate by a *free* arborisation in the interior of the glomeruli which form the characteristic feature of the second layer of the bulb. These spheroidal bodies were first observed by Leydig in the fish, and constitute Meynert's "stratum glomerulosum." Each glomerulus consists almost entirely of the interlacements of the olfactory fibres derived from the superficial layer on the one hand, and, upon the other, of those derived from the cells of the fourth layer; whilst a few nuclear-like cells are found within and around the glomerulus. The silver-chrome method shows that the non-medullated fibres on entering the glomerulus split up into arborisations of excessively flexuous, thickened and varicose fibrils (*Cajal*), which never again emerge from the glomerulus. Here they intertwine, but do not inosculate, with a rich arborisation of dendrites derived from a process of the cells of the fourth layer—the so-called "mitre cell." Immediately beneath the glomerular layer is a stratum of finely granular material, in which are imbedded small fusiform nerve cells which throw off a coarse protoplasmic process (dendron) towards the glomerulus, in the interior of which it loses itself as a dendritic arborisation. An extremely fine axon passes from the cell deeply towards the granule layer of the bulb and, bending at right angles, passes backwards amongst the medullated fibres of the tract.

Layer of Mitre Cells.—Between the preceding and the deepest layer of the bulb lies a stratum of cells, remarkable for their size, configuration, and relationships. These elements are usually of large size, of mitre-like form, throw off lateral processes ramifying through the neighbouring molecular layer, whilst, from their lower aspect descends a coarse dendron, which, entering a glomerulus, breaks into a terminal ramification of dendrites interlacing with the olfactory nerve fibres, as already noted. From the deep aspect of the mitre cells a stout axon passes upwards to the granule layer, and here, bending backwards, becomes continuous with a medullated fibre of the olfactory tract, for distribution to the cerebral cortex. In this course through the granule layer, the axon throws downwards several vertical collaterals, which ramify in the molecular layer (*Van Gehuchten* and *Martin*).

Layer of Granules and Medullated Fibres.—This, the deepest layer of the grey matter of the bulb, is of considerable thickness, and is formed by granules similar in form to those of the cerebellar granule layer, but separated into compact groups by the passage of fasciculi of medullated fibres. According to Cajal and others they differ morphologically from the granules of the rust-coloured layer of the cerebellum; these possess a distinct axis-cylinder, while those of the olfactory bulb are devoid of such.* These spherical or angular cells throw off two processes; one, downwards to the mitre layer, where it ends in a terminal plume in close contiguity to the lateral dendrons of the mitral cells; the other, far less conspicuous, passes inwards to ramify amongst the granule groups at a deeper site. The terminal dendrites of the peripheral plume are thickly beset with minute spiny projections along their course, like the similar formations along the terminal plumules of the pyramidal cells of the cerebral cortex. The **peripheral** dendron is always present; the central dendron may be insignificant or altogether wanting (*Pedro Ramon*). Golgi has, moreover, described large sized stellate cells sparsely scattered in the granule layer, which, besides protoplasmic processes, possess an axon which (according to Cajal) always ends in a rich arborisation within the molecular layer of the bulb. The medullated fibres found in this deepest layer of the bulb consist very largely of the axons derived from the mitre and small fusiform cells; but also embrace medullated fibres which pass from the brain to the bulb (centrifugal), and end in free ramifications in the granule layer.

The Retina.—The nerve elements of the retina consist of a superficial layer—the well-known rods and cones; of an outer and inner granule layer, separated by outer and inner plexiform relational fields; and of an innermost layer of ganglionic cells and optic nerve fibres. The rods and cones of the superficial layer are morphologically continuous with the elements of the outer granule layer, but are usually, for convenience, described separately. The inner granule layer differs essentially from the outer not alone in the conformation of its constituent elements—the granule cells, but in the presence of two other nerve elements—the horizontal cells and retinal spongioblasts: whilst the outer granule layer is a comparatively simple structure—the inner is much more complex and may be subdivided into three layers:—

---

* This opinion, which has given rise to the doctrine of "amacrine cells," or nerve cells without axis-cylinders, appears to us to be satisfactorily confuted by Dr. Hill, who has discovered the axon and figured it. See "Notes on Granules," by Alex Hill, *Brain*, vol. xx., p. 125.

(*a*) Superficial horizontal cells;
(*b*) Bipolar granule cell;
(*c*) Spongioblasts of the deepest stratum.

Lastly, the inner plexiform or relational field is one of very great complexity, embracing, as it does, the ramifying branches of the bipolar granule cells, the dendrites of the ganglionic series, and the branches of the retinal spongioblasts.

Retinal Rods.—The rods are cylindrical bodies, fine in mammals and nocturnal birds (in man 50 μ to 60 μ long by 2 μ thick, according to Max Schultze); but of large size in Batrachia, diurnal birds and fishes (*Cajal*), consisting of an inner segment or body, and an outer segment perfectly cylindrical and unpointed. Unlike the cones they are not attached directly to the body of the granule cell of the next layer, but through the medium of a peripheral process extending from the latter; so that the granule cells of the rods resemble a bipolar ganglion cell, of which the finer or centric process extends to the outer plexiform layer. Here this process ends in a small spherical swelling embraced by the dendritic peripheral branch of the cells of the deeper granule layer. The rod fibres are always finer than the cone fibres.

Retinal Cones.—The cone, always shorter than the rod, consists of a flask-shaped body or inner segment, about 6 μ in thickness, terminated in a pointed conical extremity; and an outer segment, which is more strongly refractile than the inner segment. Schultze has described a longitudinal striation of the outer segment of both rods and cones in fishes, amphibia, and mammals, including man; and likewise a similar striation of the inner segments in man and mammals. The body is distinctly continuous with the granule cell of the cone—an ovoid body with large nucleus, from which a centric process descends to the outer reticulated or plexiform layer, where it ends in a conical swelling with a few lateral free fibres. In reptiles the rods are absent, the cones alone being found. It is interesting to note that Krause has shown that the rods and cones both persist after section of the optic nerves, their appearance suggesting no degenerative change. The external limiting membrane forms a natural boundary between the rods and cones and their subjacent granule cells.

Outer Plexiform Layer.—In this layer the protoplasmic branchings from the peripheral ends of the bipolar cells take part—some interlacing with the termini of the centric branch of the cone cell; others receiving between them the spherical globules of the centric branch of the rods. Ramifying fibres extend also into this layer from the horizontal cells of the subjacent stratum.

Plate IX

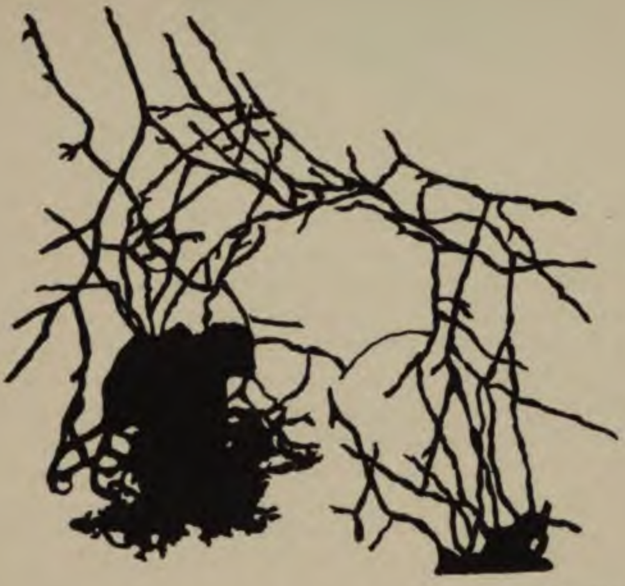


FIG. 1.

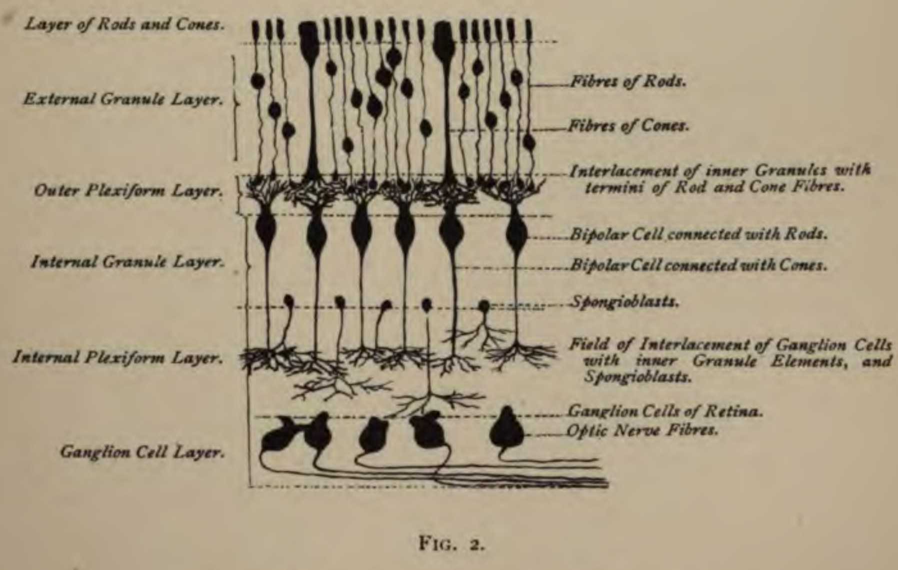

Layer of Rods and Cones.
External Granule Layer.
Outer Plexiform Layer.
Internal Granule Layer.
Internal Plexiform Layer.
Ganglion Cell Layer.
Fibres of Rods.
Fibres of Cones.
Interlacement of inner Granules with termini of Rod and Cone Fibres.
Bipolar Cell connected with Rods.
Bipolar Cell connected with Cones.
Spongioblasts.
Field of Interlacement of Ganglion Cells with inner Granule Elements, and Spongioblasts.
Ganglion Cells of Retina.
Optic Nerve Fibres.

FIG. 2.

SCHEME OF RETINAL LAYERS IN MAMMALS.

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Inner Granule Layer.—Three subdivisions are described by Cajal—(*a*) horizontal cells; (*b*) bipolar granules; (*c*) retinal spongioblasts. The former consist of small and large horizontally disposed nerve cells, of which the large-sized are the most internal; both throw off numerous protoplasmic processes and a long axon; the axon from the smaller and more superficial cell is very fine, giving origin to several short ramifying collaterals. In the deeper cells this axon is much larger, and terminates, after a lengthened course, in a rich arborisation in connection with the spherules of the rods (*Cajal*). These ramifications add, therefore, very materially to the plexiform meshwork of the third layer. The bipolar cells of this layer are fusiform in contour, give off from their peripheral pole a tuft of protoplasmic branches, which spread laterally into the outer plexiform layer; whilst the fine centric branch descends to different levels of the inner plexiform layer, ending in a plume of free branches in close relationship to the ascending dendrites of the ganglionic cells. Cajal specially distinguishes these bipolar cells into such as have a peripheral ascending plume of branches, and those with a flattened plume: the former being distributed to the spherules of the rods; the latter to the branches of the cones. The terminal arborisations of the centric or descending branch of these bipolar cells meet the dendrites of the ganglionic cells at different levels of the inner plexiform layer, thus dividing it into five or more strata.

Retinal Spongioblasts.—These cells are remarkable in the fact that they possess no axon. They are disposed in the lowermost stratum of the inner granule layer, in immediate contact with the inner plexiform layer and all their processes are directed centrally. Schäfer doubts their nervous nature.*

Inner Plexiform Layer.—We have, therefore, immediately beneath the inner granule layer, a very rich relational field, in which three distinct retinal elements are brought into immediate apposition, viz.:—

(*a*) Bipolar granule elements; (*b*) Retinal spongioblasts;
(*c*) Ganglionic cells.

This elaborate system of fibres is known as the inner plexiform layer.

Ganglionic Cells.—These cells, like those of other parts of the nervous centres, vary much in size, from 15 μ to 30 μ in diameter in the fresh state; they are flask-shaped or ovoid in contour, usually devoid of pigment, and possess a large nucleus with a prominent nucleolus. They give origin to a peripheral and centric process—the former directed into the depths of the inner plexiform layer, as a dendritic arborisation, extending laterally in the horizontal plane as far as one or other of the five stratifications of this layer, where they blend with the corresponding terminal fibres of the retinal spongioblasts and the bipolar granule elements. The centric process is really its axon, which becomes continuous, as an optic nerve fibre, with the deepest layer as it courses towards the optic nerve.

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* "The Nerve Cell considered as the Basis of Neurology," *Brain*, 1893, p. 138.

Cajal divides these ganglionic cells into three series: those whose dendrites are restricted to one stratification only; those distributed to two or more; and those which observe no such law of stratification, but are equally distributed throughout the whole depth of this plexiform layer ("cellules unistratifiées, multistratifiées, et diffuses"). So far, therefore, from the optic nerve fibres being in direct continuity with the special visual elements of the retina—the rods and cones—we find, according to the researches of Cajal, Van Gehuchten, and others, that there are two breaks in this course: the first, betwixt the dendrites of the ganglionic cells and the bipolar elements; the next, betwixt the peripheral offshoots of the latter and the termini of the centric branches of the rods and cones respectively. This is in accord with the results of physiological and pathological teaching, for we know that section of the optic nerve fibres entails fatty degeneration of the ganglion cells of the retina (*Krause*), whilst the rods and cones escape intact; moreover, in blindness from optic nerve atrophy and in glaucoma from intraocular pressure the ganglion cells suffer in like manner.

The Cerebellar Cortex—*Lamination.*—The cerebellar cortex may be regarded as two-laminated, a superficial or external, commonly termed "the molecular layer," and an internal or granule layer; whilst upon the confines of both is a series of voluminous cells peculiar to the cerebellar structure, termed the "cells of Purkinje." The leaflets or foliola of the cerebellum are mostly disposed transversely to the antero-posterior axis of the brain, and sections may be taken either in the direction of the plane of these lamellæ—*i.e.*, **frontal** sections, or across their plane antero-posteriorly—*i.e.*, **sagittal** sections. The microscopic appearance is very different in these two cases. In the antero-posterior or sagittal section, the dendritic expansions of the cells of Purkinje attain their maximum development and are displayed to the greatest advantage, whilst the nerve cells peculiar to the superficial layer, with their neuraxons, are also seen following the same plane. In frontal sections, on the other hand, the cells of Purkinje are seen as it were *in profile*, their dendritic branchings, insignificant and flattened laterally, would indicate a somewhat fan-shaped conformation in the direction of the sagittal plane. At the same time, we lose the characteristic branchings of the nerve cells peculiar to the molecular layer, and, in lieu thereof, we see a horizontal striation of this layer, due to bifurcation of the axons of the granule cells of the deeper layer, and which, being cut across in

sagittal sections, appear simply as innumerable dots scattered throughout the field at this level.

Superficial Layer of Cortex.—The external layer immediately beneath the pia has, in the embryo, a structure quite peculiar to itself, of which, however, all trace is lost in later life; it forms a peripheral zone (not as yet encroached upon by the dendrons of the cells of Purkinje) and consists, according to Cajal, of—

(1) A superficial series of granule-like cells placed vertically to the surface;

(2) A deeper layer of horizontally disposed bipolar cells lying next to the molecular layer.

In the fully developed cortex, however, the elements demanding attention are :—(*a*) The intrinsic nerve cells or elements peculiar to this layer; (*b*) the dendrons and collaterals of the cells of Purkinje, together with the nerve fibres extending from underlying structures; (*c*) non-nervous or connective elements.

(*a*) *Nerve Cells of the Superficial Layer.*—These are irregular, stellate, or polyhedral cells, most numerous in the deeper realm of this layer, having their long axis disposed antero-posteriorly—*i.e.*, in the plane of the dendrons of the cells of Purkinje. Numerous long protoplasmic processes are given off by these cells; but, in particular, a fine and greatly elongated axon, which also courses along the plane of these same dendrons. Along this course, which is more or less parallel to the surface of the cortex, vertical offshoots or collaterals descend to the cells of Purkinje, and, becoming coarser near these cells, split up into a terminal ramification, which forms a basket-work* embracing the body of the latter as far as the commencement of its axon. Contracted over the lower pole of the cell, this basket-work extends brush-like a short distance along the neuraxon, where it is still devoid of a medullary sheath. It is remarkable how this terminal basket-work escapes staining in aniline preparations, where the cells of Purkinje are yet admirably displayed. The cell with enclosing basket-work is named by Kölliker a "basket cell" (*Korbzellen*).

Close to the pial surface of the cortex a few stellate cells, much smaller, are found; their protoplasmic processes are richly developed, and the axon, which runs antero-posteriorly, ramifies extensively, but its destination is unknown.

(*b*) *Cells of Purkinje.*—These flask-shaped cells, so characteristic of the cerebellar cortex, throw off from their upper or outer pole a single or double dendron (in the latter case, giving the cell a somewhat horned appearance), from which arises a luxuriant arborisation by frequent dichotomous division as far as the surface of the cortex. These dendrites neither anastomose with each other nor with the neighbouring cells; they are strictly terminal and often turn back upon themselves. The dendritic system is spread out along the antero-posterior plane, and the branches are, moreover, thickly studded with minute thorny processes like the corresponding projections (*épines*) upon nerve cells in other regions (*Van Gehuchten, Cajal, Retzius*). From the lower or internal pole of the cell descends the neuraxon, which, passing through the granule layer of the cerebellum (becoming medullated in this course), enters the central medullated core of the lamina, probably passing out of the cerebellum to distant centres. Shortly after its origin the axon throws off some two or three delicate collaterals, which, passing upwards into the peripheral layer, end by a terminal ramification in contact with the lower dendrites of the neighbouring cells of Purkinje; thus, according to Cajal, they ensure a certain degree of functional solidarity.

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* *Endkoerben, Faserkorbe, Faserpinsel* of Kölliker.

Besides the structures just described the molecular layer displays in frontal sections (*i.e.*, sections taken *along the course of the laminæ*) a distinct striation due to fibres running at all depths parallel to the surface, which, on close examination, are seen to arise in all cases from the T-shaped bifurcation of numerous vertical fibres passing upwards into this layer from the stratum of granules below. These delicate fibres, which are really the axons of the granules of the cerebellum, give off no collaterals, pass horizontally through the dendritic expansions of the cells of Purkinje, and thus bring the granule layer into intimate connection with those arborisations. They are stated by Cajal to end after a lengthened course in a free and varicose thickening near the white matter of the lamina. If we refer to frontal sections as the "plane of lamination," and to the sagittal sections across a lamina as the "plane of arborisation," *i.e.*, of the dendrites of Purkinje, then it may be stated that the cells of Purkinje are brought into intimate functional relationship along the plane of arborisation by the axons of the cells of the peripheral layer, and the basket-work resulting from their collaterals; whilst they are bound in functional relationship along the laminar plane by the parallel T-shaped axons arising from the granules of the second layer; and at the same time their functional co-operation may be established by the recurrent collaterals arising from their axons, and ramifying in the proximity of the lower dendrites of the neighbouring cells of Purkinje.

A further relationship, however, is established between the cells of Purkinje and certain coarse fibres ("fibres grimpantes" of Cajal) which ascend from the central medulla through the granule layer, and form a dense plexus upon the primary and secondary dendrites of these cells, just as the nerve cells of the peripheral layer form the basket-work enveloping the body of the cell. The peripheral origin of these fibres is unknown.

(c) *Non-nervous or Connective Elements.*—These are of two kinds :—

(1) A large irregularly shaped dendritic cell, lying in close proximity to the cells of Purkinje, which throws off, in the granule layer, a number of thick short processes and several long vertical branches. The latter pass upwards into the peripheral layer, each fibre ending immediately beneath the pia in a small triangular or conical thickening ; these fibres have long been known as the fibres of Bergmann.

(2) Deeper in the granule layer and extending into the medullated centres are numerous stellate cells in no wise differing from the stellate glia cells common to the nervous centres at large.

Granule Layer of the Cortex.—Beneath the peripheral layer is a compact stratum of small, nearly spherical nerve cells, possessing very little protoplasm, and throwing off on all sides some few short processes, which end in miniature arborisations around the bodies of the neighbouring granules. From the cell body or one of its processes arises an extremely delicate axon, which passes vertically upwards, and, at varying levels in the outer zone, bifurcates into the T-shaped fibres which have already been alluded to in this stratum of the cortex.

Golgi and Cajal also describe large stellate cells as occurring sparsely in the granule layer, with extensive protoplasmic processes spreading in all directions—often far into the peripheral layer, and with an axon which, passing downwards into the granule layer, ramifies to an extraordinary extent amongst these granules, ending, according to Cajal, by free varicose extremities.

Moss Fibres of the Cerebellum.—In the granule layer certain notable fibres, first described by Cajal as moss fibres, appear ; they ascend from the medulla as coarse medullated fibres which, on entering the granule layer, split up and ramify extensively among the granules, presenting here and there along their course peculiar rough knotty thickenings or moss-like growth, and do not extend beyond the granule layer. These fibres are presumed to bring the granules of the cerebellum into relationship with distant nerve centres, and are suggested as the possible central nerve termini of the cerebellar tract (*Cajal*).

Our review of the foregoing types of cortical lamination in the mammalian brain prepares the way for certain deductions which have an important bearing upon the physiology and pathology of the cerebrum. In the first place, let us note that the **simpler forms of cortex** are confined to the lower margin of the cortical envelope, where it folds round the cerebral peduncle at the base—the **cornu Ammonis,** the **lower limbic lobe ("outer olfactory type"),** and also the **olfactory area** of Gratiolet.

The more **complex form of cortex,** however, spreads over the **upper limbic arc** and the whole of the **extra limbic region** of the hemisphere. It is these more complex forms of cortex which concern us chiefly; they comprise in man the extensive areas at the vertex and the whole convoluted surface of the hemispheres, as seen from above. Now, in studying the small brain of the rodent and higher animals, we find structural modifications in the cortex of this region, which appear to foreshadow the divergences observed in man. Thus, if we examine successively the cortex at different points from within outwards in a vertical section through the hemisphere, passing through the Sylvian depression, we find that—

(*a*) The first layer of the cortex is deepest at the sagittal border, and steadily diminishes in depth as we proceed outwards towards the limbic fissure;

(*b*) The second layer of small pyramidal cells *increases rapidly in depth and in wealth of cell-structure* in a reverse direction—*i.e.*, from within outwards;

(*c*) The ganglionic series of cells (which assume thick clustered nests in the upper limbic arc and over the vertex bordering on the sagittal fissure), gradually loses its confluent tendency and becomes spread out in isolated units ("solitary type") as we approach the limbic fissure externally. On the other hand, if we examine similarly a vertical section taken through the posterior moiety of the upper limbic arc (Plate vi.) we find that—

(*d*) The intercalated series of granule cells increases in richness of elements and depth of formation as we proceed outwards to the lateral aspects of the hemispheres, and backwards to the occipital pole; and reaching the limbic fissure terminates abruptly, whilst the other layers pass on uninterruptedly. If we now examine vertical sections of the hemisphere in the antero-posterior plane, we find that—

(*e*) The outer layer (peripheral zone) progressively diminishes in depth from the frontal to the occipital pole;

(*f*) The small pyramidal cells of the second layer diminish in size in the same direction;

(*g*) The granule or angular cells intercalated in the five-laminated cortex increase in richness conspicuously towards the occipital pole;

(*h*) Lastly, the ganglionic series, which near the frontal pole forms a deep layer rich in cell elements, thins out considerably backwards into a laminar or "solitary" formation; but, at the extreme occipital pole, these cells again form a somewhat deep belt with granule cells superimposed.

The obvious deductions to be made from the foregoing are that certain elements preponderate in certain fixed areas of the cortex, and that the development of certain layers appears to exclude that of another series. Thus the frontal pole and frontal extremity of the upper limbic arc are especially characterised by the preponderance of the **ganglionic series,** which accumulates here in rich clustered groups; towards the Sylvian border this element is insignificant, and it is the **small pyramidal** layer which here prevails. Towards the occipital pole mesially ("modified upper limbic type") the **granule cell** attains like importance from its notable wealth of elements and its more or less complete exclusion of the small pyramidal series; whilst outside this formation, in the extra limbic cortex, the intercalated granule belt is a notable feature, accompanying a corresponding impoverishment of the small pyramidal and ganglionic series.

A certain relationship also would seem to exist between the depth of the first layer or peripheral zone and the ganglionic series of cells; since it notably diminishes in depth as these elements thin out into the solitary type of arrangement, and this despite the marked increase in the small pyramidal series above. This mutual dependence seems to us explained by the fact that the apical processes of these large elements pass up into, and terminate in, this peripheral zone, so that any regional difference in the depth of the outer layer will be dependent on the greater or less development of these ganglionic cells. It must be borne in mind, however, that the *average* depth of the first layer increases in lower mammals and becomes shallower as we rise to the more highly organised brains—a fact which does not militate, as might at first appear, against the preceding conclusion. In the lower mammals, the absolute and relative increase in the depth of this outer layer probably means a large preponderance of the connective over the nervous element.*

Regional Distribution of the Ganglionic Cell.—Attention was first directed to the peculiarly clustered arrangement of these cells in the cortex of man and the higher apes by Professor Betz,† who denominated them "giant pyramids," and suggested their probable *motor* signification from their form, arrangement, and connections. Subsequent research appears fully to confirm the conclusion arrived at by Betz,‡ and it becomes, therefore, important to indicate the regional distribution of these elements. We find that this series of cells in man and the higher mammals (Pig, Sheep, Dog, Cat, Ape, and Man) assumes in separate regions of the cortex a different arrangement, which we have termed the **clustered** or **nested** and the **laminar** or **solitary** arrangement *—the former showing these large cells aggregated into distinct oval clusters stationed at intervals apart—the latter approaching the arrangement of these cells universally met with at the base of a sulcus, viz. :—solitary cells, stationed like sentinels wide apart, showing no tendency to grouping beyond two or three at most in certain exceptional areas. In lower mammals (Rabbit and Rat), these discrete or distant clusters do not appear; but what we take to be the homologue of this series forms **confluent groups**—the nested arrangement being scarcely indicated, and a deep and dense formation replacing the latter. As already observed, however, these confluent groups thin out, in certain regions, into linear file, assuming the laminar or solitary arrangement. The cells of this series in these lowly-organised brains are peculiar in their extremely elongate pyramidal or fusiform contour, and approach in this respect the form of the *larger pyramids* in the human cortex rather than the configuration of the *motor* cell. As we pass from the confluent groups of elongated elements in the Rodent to the more specialised areas of higher mammals, we find that—

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* See upon this point, Meynert, "Brain of Mammals," *Syd. Soc.*, p. 383; also, *Brain*, vol. i., p. 358.

† "Anatomischer Nachweis zweier Gehirncentra," Prof. Betz, *Centralblatt f. d. Med. Wissensch.*, Aug., 1884.

‡ It is true that Meynert would dispose of the assumed significance of these cells on the ground that their large size depends on the distance which their apical process has to traverse in reaching the outer layer, and their gradual increase in dimensions being, as he states, proportionate to this distance. The "gradual increase in size" alluded to proves to us that Meynert has failed to identify the elements referred to—probably mistaking for them the larger pyramids; and finally his argument falls to the ground when it is seen that the second layer of the "modified lower limbic type" contains larger elements than any of its subjacent layers.

(1) The cells become less elongate, more swollen, and irregular in contour;

(2) Their groupings become more and more discrete;

(3) The individual groups grow larger in size;

(4) The clustered arrangement occupies a wider range of cortex.

In Plates i. and v. this series of cells is richly represented; they are densely congregated towards the margin of the hemisphere, and thence, continued to the limbic fissure, occupy the whole area embraced by Nos. 7 and 9 in Ferrier's work.† Further back, however, this layer diminishes in depth and in wealth of cells, except at the exposed margin of the hemisphere, where it still remains a rich formation; beyond the margin and over the extra-limbic region, as far as the limbic fissure, the cells rapidly thin out into a simple linear series, and the five-laminated cortex appears. Still further back the series, in like manner, thins out into a mere insignificant formation—yet always most richly developed along the sagittal margin of the hemisphere.

Plate v., fig. 1, represents the arrangement of the ganglionic series in the pig, the regional distribution of which is almost identical in formation with that of the sheep. For both these animals, it may be stated that a *five-laminated* cortex, with *clustered cell-groups*, spreads over the anterior half of the upper limbic arc (which in these animals becomes superficial on the upper aspect of the hemisphere) over the frontal pole and along the first (or Sylvian) and second parietal convolution. Between these tracts is embraced the area of the third and fourth parietal convolutions, which have a *six-laminated cortex* and a distinctly *solitary arrangement* of these cells.

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* "Comparative Structure of Cortex Cerebri," *Trans. Roy. Soc.*, part i., 1880.

† *Functions of the Brain*, second edition, p. 259, fig. 78.

If we examine the regional distribution in the cat, the anterior portion of the upper limbic arc in front of and above the crucial sulcus; the frontal lobe; the first parietal, or Sylvian; and the anterior extremity of the fourth parietal or sagittal convolution, will all be found to exhibit the laminated cortex and nested cells; yet the formation, excessively rich in the sigmoid gyri around the crucial sulcus, becomes much poorer in other regions. The six-laminated type extends over the whole extent of the upper limbic arc, behind the crucial sulcus, as far back as the retro-limbic annectant.

The distribution of these nested groups of ganglionic cells in the ocelot, reproduces, in fact, very nearly the arrangement met with in the cat.

The distribution of the same formation in the Barbary ape foreshadows the arrangement which pertains to the more highly-developed cortex of man.

It will be observed from the foregoing remarks that the **crucial sulcus** in all these animals forms a distinct limit to two types of lamination—peculiar to the vertex—the **five-** and the **six-laminated types,** and that this distinction is continued upon the mesial aspect of the hemisphere into which this sulcus extends; that, similarly, at the frontal pole of the hemisphere, the vertical sulcus, regarded by Broca as the representative of the fissure of Rolando, also separates an inner or five-laminated from an outer or six-laminated cortex; whilst the first parietal or Sylvian convolution in the pig and sheep partakes, in front of the Sylvian fissure, of the five-laminated type.

In 1882,* after a minute enquiry into the cortical envelope of the brain in mammals, the author had reason to express himself as follows:—"The more fully I investigate the minute structure of the cortex and its deep connections, the more forcibly am I impressed with the belief that the various fissures and sulci are not mere accidental productions,† but have a deep significance of their own, dividing off the cortical superficies into *morphologically*, if not *physiologically*, *distinct organs*. Hitherto the fissures and sulci which I have found to be boundary lines of distinct cortical realms are the following:—

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* *Op. cit.*, p. 724.

† That is, the result of pressure merely during the development of the cranial arch.

"(1) The limbic fissure.
(2) The infra-parietal sulcus.
(3) The crucial sulcus.
(4) The superior parietal sulcus.
(5) The inter-parietal sulcus.
(6) The olfactory sulcus.
(7) The fissure of Rolando."

Contrasts between the Brain of Man and of Lower Mammals.—When we contrast the cortex of the human brain and of the ape with that of the mammalian series below these types, certain strongly-marked resemblances in intimate structure, as well as equally notable divergences, present themselves. With respect first to the **resemblances,** it is to be noted that the various types of cortical lamination described in the lower mammals are reproduced in the brain of the ape and man; and that the several layers maintain the same relative position throughout their depth, except where in certain cases a layer is wanting, or a new layer is interposed. Again, the individual elements constituting these layers—the granule cell, the angular cell, the spindle, the pyramidal element—although differing somewhat in dimensions and general contour, are yet sufficiently alike for their identification apart from their mere position in the cortex.

In the next place, the lower limbic margin of the cortical envelope always presents the simpler forms of cortex; while, towards the vertex and mesially both towards frontal and occipital poles, the more complex forms of cortex prevail. Another striking resemblance occurs in the distribution of these laminar types—that characterised by the **granule cell** predominating towards the **occipital pole;** that of the **five-laminated type** being especially developed towards the **frontal pole:** with this there is associated finally the gradual diminution in size of the one element towards the temporal and occipital lobe, and the increased dimensions and richness of formation of the other element in the same direction. These are some of the more striking resemblances presented between the cortex cerebri of man and that of the lower mammals.

As to the **divergences** presented by these structures, we are early struck by the fact that the **abruptness of transition** from one to another type of cortex, seen, *e.g.*, in the rodent, is not a feature in the human brain; in fact, transition-realms invariably intervene betwixt different types of lamination. The one fades into the other form so gradually that a line of demarcation can rarely be drawn. Thus, the five-laminated cortex characterising the "motor area" of the human brain affords no abrupt transition into the six-laminated cortex lying external and posterior to it; a mixed type intervenes, to which we have applied the term of "transition-realm."

In the second place, the cells which we have ventured to term "motor" in the fourth layer of the human cortex, differ from what we

Plate XI.

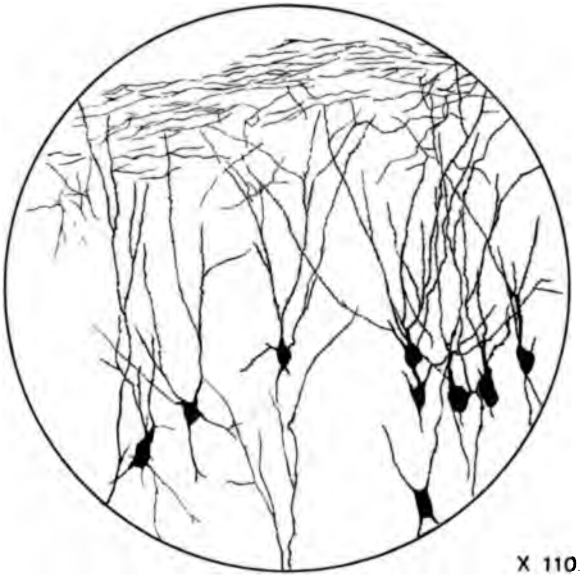

FIG. 1.

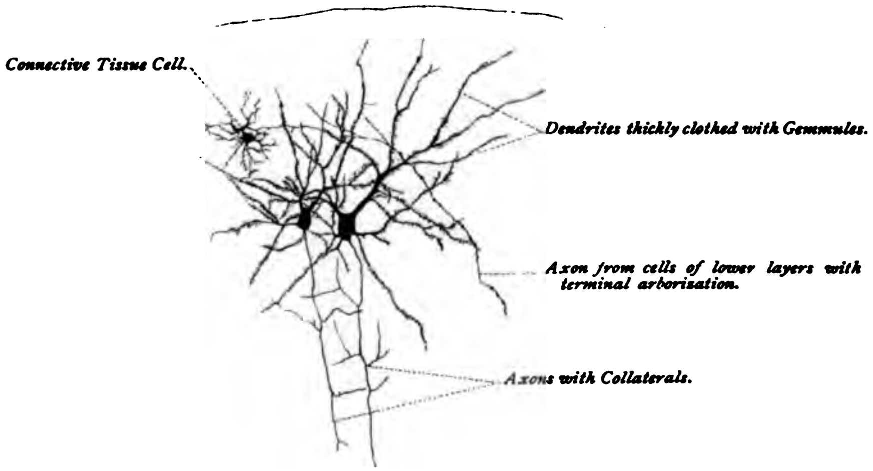
Connective Tissue Cell.
Dendrites thickly clothed with Gemmules.
Axon from cells of lower layers with terminal arborization.
Axons with Collaterals.
X 110

FIG. 2.

BRAIN OF YOUNG RAT: SECOND LAYER OF CORTEX NERVE-CELLS, SHOWING EXTREMELY HIRSUTE DENDRITES AND DESCENDING AXONS.

Bale, Sons & Danielsson, Ltd. Lith.

have regarded as the homologous series in lower mammals, in being restricted as a typical formation to a comparatively limited area of the cortex—that of the rodent, *e.g.*, being spread over a far wider proportionate area of the hemisphere. This concentration of these cell-groups is best seen in carnivora, where, as already shown, they crowd around the crucial sulcus, especially at the angle of the sigmoid gyrus. They exhibit the tendency in a less marked degree in the higher apes, whilst in man they are concentrated in three or four districts occupying, as before stated, but a comparatively limited area. A still more notable distinction between the higher and lower forms of brain presented by this formation, is the nested arrangement observed by Betz in the human brain. This segregation is complete, the groups being large and far apart. As we descend the scale, however, the more do we observe the tendency for such groups to become confluent, and the series to be disposed as an equable stratum.

Lamination of the Motor Area in Man.—That region of the cortex which has been shown in animals to be electrically excitable, and which upon stimulation calls forth responsive movements, has been termed the "motor area." It is, as we have just seen, characterised by a highly specialised structural arrangement. It is all the more essential that its structure in man should be clearly defined here, since it has been the subject of dispute between such writers as Meynert, Betz, Baillarger, Mierzejewski, and others, some authorities speaking of it as a *five-laminated* and others as a *six-laminated* type. At the outset, therefore, it is well to define our own view of the case, which is briefly as follows:—The cortex *typical* of motor areas is a **five-laminated formation,** and the more absolutely the granule-cell formation (which, when intercalated, gives us the six-laminated type) is excluded, the more highly specialised become those groups of enormous nerve cells which go by the name of the "nests" of Betz. Where, therefore, these cell-clusters are best represented, there we find a five-laminated, *not* a six-laminated, cortex; in other words, at these sites the granule-cell layer no longer exists. Such a specialised cortex is not spread uniformly over a large convolutionary surface at the vertex—any such notion would be very far from correct; but it occupies very irregular, limited, and unequal areas along the course of the ascending frontal and the junctions between it and the frontal gyri, as well as the "paracentral lobule." These positions we shall more clearly define later on.

Such irregularly-disposed areas are severed from each other by a transitional form of lamination, whereby these districts gradually merge into the six-laminated cortex surrounding them. This highly-specialised cortical formation is constituted as follows:—

First Layer.—An extremely delicate pale zone limits the cortex externally; it presents all the features already described as peculiar to the cortical neuroglia (see p. 94). The outer surface, upon which the intima pia rests, presents numerous flattened cells, from which excessively delicate processes pass downwards into this layer. These cellular elements are often found, detached from fresh sections, floating in the medium around; they form, in fact, a kind of epithelial limiting layer, extremely delicate and translucent. This first layer, or peripheral zone, exhibits a pellucid homogeneous matrix (becoming finely molecular with reagents) and three structural constituents—(*a*) non-nervous, (*b*) nervous, (*c*) vascular.

(*a*) The *non-nervous constituents* are not numerous, are widely dispersed, and belong to the two categories of the perivascular or adventitial elements and the elements of the lymph-connective system already referred to (p. 94). The former measure 6 μ to 9 μ in diameter, possess a spheroidal nucleus, stain well, and are seen disposed along the course of the blood-vessels. The latter often measure 13 μ in diameter, possess one and occasionally two or three nuclei, are spheroidal, flask-shaped, or irregular in contour, stain uniformly and very faintly, and throw off numerous excessively delicate processes, which in healthy fresh cortex can only be distinguished with difficulty.

(*b*) The *nervous constituents* embrace a series of medullated, non-medullated nerve fibres, and nerve cells peculiar to this layer.

The medullated nerve fibres course along the outer division of this zonular layer, in a horizontal direction, lying parallel to the pial surface of the cortex; many of these are observed to pass downwards into the deeper layers of the grey matter. In certain regions, this medullated tract or tangential belt, so characteristic of the first cortical layer, lies at a somewhat deeper level near the second layer of cells. In most small mammals these fibres take also an antero-posterior direction. The non-medullated nerve fibres are of two kinds, viz., protoplasmic processes or dendrons from the underlying cells (p. 128); and terminal distribution of axons from the cells peculiar to this layer, and also from those of subjacent tracts.

The nerve cells, very sparse in number, are chiefly limited to the lower two-thirds of the peripheral zone. Three kinds of nerve cells are described by Cajal:—the polygonal or stellate, with numerous protoplasmic processes, but with *one* axon only, which, arising from the body of the cell, ramifies very extensively in all directions in this zone, terminating in fine, varicose and free fibrils; long fusiform, bipolar cells disposed antero-posteriorly, and, from either pole, giving origin to an exceedingly long protoplasmic process, which along its course throws off from time to time collateral branches upwards to the pial surface. The peculiar feature, however, in these cells is the *plurality* of their axons; *each* main protoplasmic branch appears continuous with, or gives off at some distance from the cell, an axon which, coursing horizontally, throws off numerous ascending collaterals, ending in extremely delicate branches throughout a very extensive region of this layer. Such axons are probably medullated (*Cajal*). Supernumerary axons arise also from the secondary protoplasmic branches, usually ascending in their course. These cells, therefore, exhibit the unusual feature of protoplasmic processes ending in axons.

Lastly, there are triangular elements which usually throw off three main protoplasmic processes; two of which run horizontally to the surface, or course somewhat obliquely upwards; whilst the third passes downwards to bifurcate into arcuate processes, from which arise two, three, or more axons probably medullated, and of extensive distribution. A smaller and more rounded element is also found here, which not only originates axons from its protoplasmic processes, but sends off an axon from the cell body, from which numerous collaterals arise and ascend to the surface.

In all these nerve cells of the peripheral zone, besides the plurality of axons, their extensive ramification, and their origin from protoplasmic processes, the characteristic features are the great paucity of dendritic branchings, the great length of the dendrons, and the absence of varicosities and collateral spines (*Cajal*).

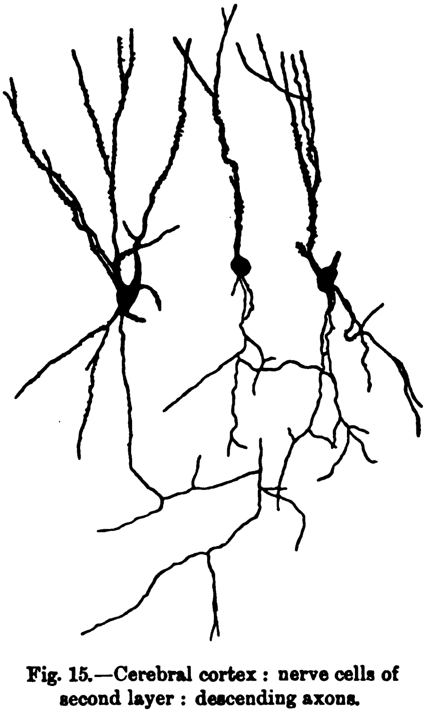

Fig. 15.—Cerebral cortex: nerve cells of second layer: descending axons.

(*c*) The *vascular elements* pass as long straight vessels for deep distribution, and as short branched and smaller vessels through its structure; they call for no special remark here.

Second Layer.—A narrow belt of very closely aggregated nerve cells of irregular marginal contour, oval, pyramidal, or angular, with a proportionately large nucleus, forms this stratum. The cells vary much in size, and, as we have previously remarked, are much more richly developed in some than in other regions of the brain. They measure (fig. 15) from 11 μ to 23 μ in length, 6 μ to 9 μ in breadth, the nucleus being often 6 μ in diameter.

They exhibit numerous delicate processes, radiating from the base and sides; but a distinct apical process or frequently a bi-corned apex passes up radially to the surface of the cortex and undergoes rapid subdivision. In the cortex at the vertex in a rat and rabbit this second layer is practically absent; although it may be traced as small, scattered, appressed groupings of cells on the lateral aspects of the brain. To see these cells to advantage in such brains we must examine the lower arc of the limbic lobe. The same may be said for the sheep; but, in the pig, this layer is a fairly notable one throughout, reaching a thickness of 138 μ. In the cat, dog, and man the depth attained may be 279 μ.

Third Layer.—Subjacent to the above lies a deep belt of nerve cells, the elements of which are characterised by their more or less elongated or pyramidal contour, and by the tendency to gradual increase in their size as they lie deeper in the cortex. The summit of these cells is elongated into a long delicate apex process, which passes radially upwards towards the peripheral zone. The opposite pole of the cell is irregularly dentated by the extension of numerous delicate processes, which are thrown off from the cell in all directions around: none of these processes turn upwards and pursue the course of the apex process. The dimensions of these cells in the outermost zone average 12 μ × 8 μ; those of the deeper regions of this layer 22 μ up to even 41 μ in length, and 23 μ in shorter diameter. Each cell possesses a large nucleus and a distinct nucleolus. Small pyramidal cells, however, no larger than those at the commencement of this layer, occur even at the deepest part, side by side with the largest.

Fourth Layer.—This layer presents us with the highly characteristic nerve element which we have already dealt with under the name of "motor cell." These great elements are found modified in different cortical realms as follows:—

(*a*) *In the highest regions* of the motor area (summit of central gyri and paracentral lobule) they are not only of gigantic size, as compared with other nerve cells around, but they form here the large clusters recognised by Betz.

(*b*) *In the lowest regions* of the motor area (lower end of central and junction with third frontal gyrus) they become small in size, even less than the superjacent elements of the third layer, but still retain their clustered disposition.

(*c*) Towards every sulcus these cells, be they large or small, lose their groupings, and at the base of the sulcus they always assume the drawn-out single file, spoken of as the "solitary" type of arrangement.

(*d*) Lastly, as this laminar type passes into that of the sensory realms, these cells have superimposed on them a layer of granule cells, but still retain a somewhat clustered disposition so characterising the transitional cortex; and they ultimately assume the solitary arrangement always seen in a sulcus, *throughout the convolution at all heights*, becoming, in fact, the six-laminated cortex typical of sensory areas. Such are the modifications undergone by these elements at different localities in the cortex.

Fifth Layer.—This layer is represented by the series of spindle cells, which, beneath the summit of a convolution, are disposed radially to the surface in regular columns, separated by bundles of medullated fasciculi, ascending from the central medullated core of the gyrus. Towards a sulcus they lose this radial disposition, and at the bottom of the sulcus are disposed in a narrow belt, their long axes horizontal to the surface, a position aptly termed *reclinate* by Dr. Major. These cells measure from 25 μ to 32 μ in length, by 9 μ to 13 μ in breadth, and exhibit a large oval nucleus.

Distribution of the Motor-cell Groups.—The specialised five-

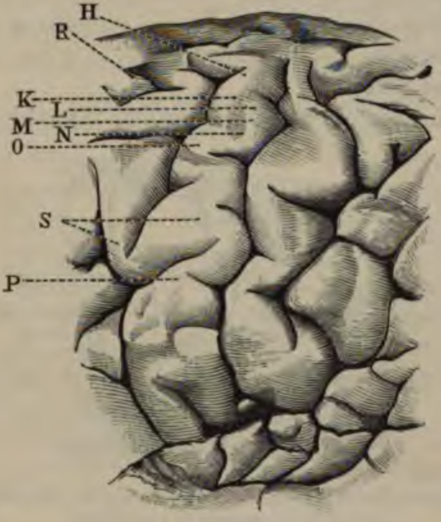

Fig. 16.—Left ascending frontal and parietal convolutions seen from the side, with the attached frontal gyri included in scheme of examination.

H-K, Third group of ganglionic cells.
N-O, Fourth group of ganglionic cells.
M-N, Barren area.
R, Region of large elongate cells.
P, Fifth group of ganglionic cells.

laminated cortex, with the cell clusters above referred to, has been stated to occupy certain areas of the ascending frontal, the three frontal gyri and the "paracentral" lobule; it remains for us to indicate more particularly the exact site occupied by this type. In the scheme now presented, the results of an investigation into the localisation of these areas in eight human brains, made in 1878 by the author in conjunction with Dr. Henry Clark, are given.* The arrangement and distribution were strangely uniform in all these cases (see figs. 16 and 17). Variations in the extent of these areas, of course, presented themselves, but not to such an extent as to vitiate the general result arrived at, viz., that such cell-clusters were grouped into several distinct areas, very clearly and definitely interrupted by the transitional type of cortex. The variations in the extent of such areas are no more than might be anticipated from the developmental variations indicated by the form of the central and neighbouring gyri. The upper end of the ascending frontal and its junction with the upper frontal gyrus are, as is well known, very variable in form and complexity, and such variations are, in our opinion, closely related to the more or less rich development of the specialised cortex under consideration. Reference to the scheme shows us that the ascending frontal gyrus may, in general, for convenience of description be considered as consisting of two segments—an upper, comprising two-thirds its length, into which run the superior and middle frontal; and a lower, comprising the remaining third, continuous with the inferior frontal in front, and with the ascending parietal behind.

Taking first the upper two-thirds, we find that the upper end has

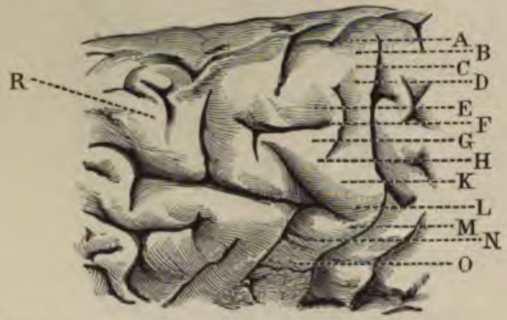

Fig. 17.—Left ascending frontal and parietal gyri, with the attached frontal convolutions, as seen at the vertex.

A–D,	First group of ganglionic cells.	M–N,	Barren area.
E–G,	Second " "	N–O,	Fourth group of ganglionic cells.
H–K,	Third " "	R,	Region of large elongate cells.

a somewhat broad attachment to the upper frontal. The lower end receives the middle frontal usually as a narrower-folded convolution, whilst between either junction a sinuous knee-like bend of the convolution exists. The broad upper extremity continuous with the upper frontal is the site of two important clustered groups (A–D and E–G); the plump lobule intervening between both upper frontals is the site of two other similar groups (H–K); lastly, the extreme posterior end of the middle frontal gyrus shows similar cell groupings (N–O), the areas of which extend into those of the ascending frontal at their lines of attachment.

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* "The Cortical Lamination of the Motor Area of the Brain," by Bevan Lewis and Henry Clarke, *Proc. Roy. Soc.*, No. 185, 1878.

The upper group (A–D) presents by far the larger cells and the more perfect and dense clusters. Such clusters occupy especially the *parietal* aspect of the convolution, which is adjacent to the ascending parietal convolution. They appear, therefore, in the cortex forming the wall of the Rolandic fissure, and creep up towards the summit, where they rapidly thin out and disappear.

The second group (E–G), connected with the lower attachment of the upper frontal, is entirely restricted to the frontal aspect of this gyrus, and does not overleap the confines of the vertex and spread into the Rolandic fissure, except at its most inferior part.

The third group (H–K) forms a large area, covering the parietal or Rolandic wall of its knee-like lobule (upper two-thirds), and spreads over the summit of the convolution at this site. Between it and the fourth group occurs a narrow territory wholly devoid of this formation; transitional cortex extending until we reach the latter group.

At the junction of the middle frontal (N–O).—This group, as before stated, becomes continuous with that of the middle frontal; it also begins with the fissure of Rolando and sweeps over the vertex.

The fifth and sixth groups (P) are indicated approximately on the scheme, but appear subject to considerable variations in extent.

To the foregoing groups must be added a further area, occupying the posterior two-thirds of the lobule on the inner or mesial aspect of the central gyri, lying in front of the fissure of Rolando and above the gyrus fornicatus, usually termed the paracentral lobule. Some enormous cells are found in the groupings of this area.

Transition-Realms of Motor Cortex.—It will be observed that, in the above enumeration of specialised areas, we have by no means covered the ground assigned to the motor area by Prof. Ferrier: the lower end of the ascending frontal, the whole of the ascending parietal, as well as the postero-parietal lobule have been omitted. In fact, these latter regions do not exhibit the specialised cortex referred to, but are covered by cortex transitional in its character between the former and what we find existing in sensory realms. If, for instance, the upper extremity of the ascending parietal be subjected to examination, we find that its interior aspect, dipping down into the Rolandic fissure, also possesses large ganglionic cells similar to those in the motor area in advance of this site. The nests or clusters, however, are not only thinly scattered, but contain few cells, and the latter diminish rapidly in size at lower levels along this convolution; it is only at the upper extremity of the gyrus that large cells are found.

Throughout by far the greater extent of this convolution, the cells of this layer are exact representatives of those found in the ascending frontal, *but are greatly diminished in size*, and although often arranged in clustered groups, *the groups are poor in elements and sparse.*

The major distinction between the transitional and specialised motor cortex is in the presence of a gradually increasing belt of small pyramidal or angular cells, which are almost identical with those of the second layer, and which here insinuate themselves between the largest cells of the third layer and the sparse nests of the ganglionic cells. Thus, with the fading-off of this rich clustered formation, we get the intercalation of an entirely new layer of elements, which grows in importance as we approach sensory realms. Now the whole ascending parietal, postero-parietal, and lower end of the ascending frontal divisions, partake of this six-laminated type of cortex; and, moreover, as we approach the margin of the brain-mantle—*i.e.*, the lower end of the central gyri—the "motor" cells become smaller and yet smaller, forming eventually insignificant clusters of minute elements. Roughly stating the case, we may say that the fissure of Rolando in the upper two-thirds of its extent, separates the typical motor cortex from the transitional cortex; whilst, in like manner, the interparietal fissure is the boundary between the transitional and the typical sensory cortex below and posterior to it.

It will be apparent from the foregoing chapter on the histological structure of the cortex cerebri that its many varieties of type depend, for the most part, upon the operation of one or more of the following circumstances. There may be—

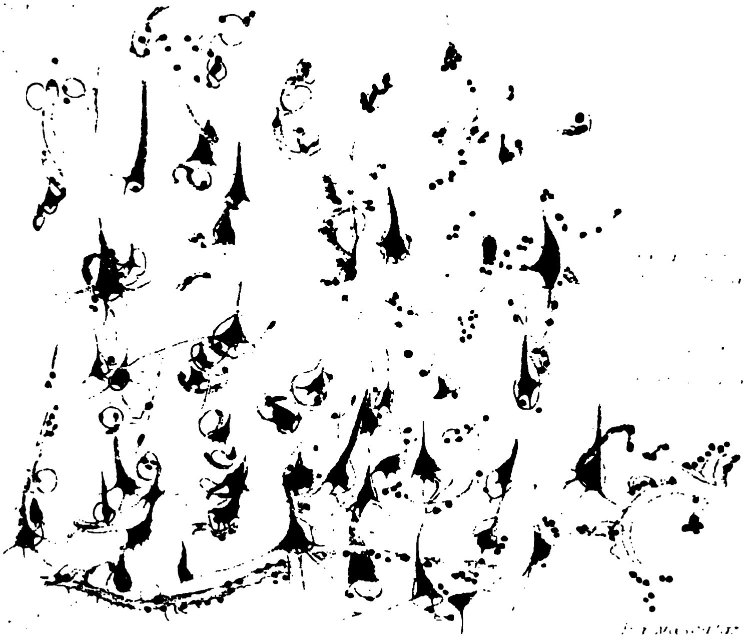
(1) **Inverse development** of superimposed layers—such, for instance, as was noted in the rodent's brain, where the third layer of cells invariably became shallower with increasing richness of the second layer of angular elements and *vice versâ*. As the one formation tends to die out, the other tends to increase in thickness and density.

(2) **Substitutional stratification** may occur—*i.e.*, a layer of cells may have other elements mixed with it, and gradually preponderating to the exclusion of its own cells, and then a change in type may occur; *e.g.*, the granule cells may gradually intermingle with the angular elements, and excluding them entirely, form a deep belt in their place, or *vice versâ*.

(3) **Intercalation of new layers,** as in the appearance of a six-laminated type, where the angular elements gradually insinuate themselves between the third layer of pyramidal cells and the subjacent ganglionic series. Or, again, an altered type of cortex may proceed from—

(4) An unusual development of the elements of a certain layer, as when the angular element of the second layer develops into the large

Plate XLI.



[illegible] Motor Cortex of brain of Cat specially prepared to show relationships between the Nerve cells & the lymphatic channels [illegible] Cortex

Bale & Danielsson Ltd. Sculp

[illegible] modified lower limbic type of [illegible] elements of the fifth layer in [illegible] larger complex cells of the [illegible] whole range of the cortex. [illegible] work in modifying its [illegible] examination to the [illegible] and its relations [illegible] variations in size [illegible] position [illegible] connections [illegible]

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20 μ	69 μ × 27 μ
17 μ	41 μ × 18 μ
41 μ	88 μ × 41 μ
37 μ	55 μ × 32 μ
× 24 μ	...

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globose cell of this layer in the **modified lower limbic type** of the rat, rabbit, mole, &c.; or where the elements of the fifth layer in the sensory cortex become changed into the larger complex cells of the **motor groupings.** As we traverse the whole range of the cortex, one or other of the above influences is at work in modifying its form of stratification.

Passing now from the question of cortical lamination to the cell itself, its conditions of life and functional activity, and its relationship to its immediate environment, let us first ask ourselves what significance, if any, is presented by the great **variations in size** of the different nerve cells: is it but an accident of their position in the cortex as to relative depth: is it indicative merely of the *age* of the cell: is it dependent on their specific functional connections: or upon the degree of complexity attained by the nerve element? Is it a mere "accident" of their position? This has been assumed by Meynert upon premises which cannot for one moment be admitted. In an article published some twenty years since,* Meynert summarily dismisses the observations of Betz on the "giant cells" of the anterior central convolution ("ascending frontal") as of no importance, because the explanation of their huge size is solely due, according to Professor Meynert, to the greater depth of the cortex of this convolution; the apex processes of these cells, therefore, having to traverse a greater distance in their low-lying groups ere they reach the outer layer of the cortex. Their groupings, also, he explains as a mere pressing together of the cells by the bundles of nerve fibres passing upwards from the medulla of the gyrus. It is obviously natural to suppose that the greater the distance along which a nerve cell has to transmit its energy, the larger will that nerve cell probably be; in the next place, as we are dealing with the *non-medullated* fibre of the apex, we might also assume that the loss by diffusion around may also demand a comparatively stronger discharge in such a case, and hence a proportionately larger cell: all this is, of course, in accordance with Meynert's assumption, if we regard the dendrites as functionating *cellulifugally;* but, the reverse is now accepted as the true course pursued by the nerve current. Moreover, that the pyramidal cells steadily increase in dimensions with their depth is also in favour of his position, *were it invariably true;* but this is not the case. It has been shown in our examination of the brain-cortex in man and in mammals, that alongside the largest pyramidal cells are numbers of others of the *smallest* dimensions, which, according to Meynert's view, should be much larger than the superimposed elements. Even in the woodcut illustrative of the five-laminated cortex given in Meynert's original memoir,* we find numerous exceptions to his rule, that the smaller element is always higher in the cortex, and, given a section of brain examined by the fresh methods, such exceptions become very numerous indeed. That the *general tendency* to this larger size with their deeper position *is* maintained, we of course allow; what we dispute is the explanation afforded, which leaves out of consideration the numerous exceptional small elements referred to.

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* *Psychiatrisches Centralblatt*, No. 6, 1878.

In the next place, were this explanation held tenable for this form of cortex, the formation described by us as the **modified lower limbic cortex** of the rodent would entirely confute such a principle, since here we have a series of **very large cells,** the largest by far in the whole depth, here lying **quite superficial** as the *second layer in the cortex.*

What then is the more probable explanation of this increase in size of the cell? If we carefully note a section of fresh brain, we find that although the majority of the pyramidal cells steadily enlarge at greater depths—the ganglionic cell-clusters, but a very short remove from the largest pyramidal cells, represent an enormous leap in dimensions. Plates i. and v. represent conclusively what we have here stated: the outlines of the cells are represented at their respective levels as sketched by the camera lucida, and it is seen that the upper elements measure but 18 μ × 11 μ, being quite superficial in the series of small pyramids; that the lowest of the series include elements measuring but 36 μ × 23 μ, although at a depth of 116 μ beneath the former: whilst a little higher we find numerous cells measuring 18 μ × 13 μ in size—*i.e.*, very slightly larger than those of the superficial series, although 953 μ beneath them. When, however, we pass from the largest pyramidal to the ganglionic cells lying only 209 μ lower down, we come suddenly upon huge elements measuring from 84 μ to 97 μ in length, by 36 μ to 46 μ in breadth. The increase in dimensions, therefore, is so sudden as to be out of all proportion to the greater depth of this layer. Is there, then, no constant relation between the size of the cell and other conditions to which it is exposed, which may give us the required explanation?

This we believe to be the case: we find as a constant accompaniment of increasing bulk, much more complex relationships with surrounding cell-districts—in other words, *the larger the cell,* the *greater the number of its branches.* But the older the nerve cell, the longer time has it had for the establishment of organised relationships around; and hence it follows that the *older cell* is also the *larger element.* In fact, it appears to us that the **size** of the **nerve cell** is chiefly dependent upon its **age** and the multiplicity of its **surrounding connections.** There is, however, another factor which must be allowed much weight in the case of the motor cortex. The medullated fibre (axon), which arises from the basal extremity of the great motor cells, traverses uninterruptedly an enormous distance to reach the respective cell-groups which represent in the spinal cord the musculature of the limbs. The distance traversed is very unequal between the lumbar and cervical groups; the cortical centres representing the lower extremities having not only a greater distance through which to discharge their energy, but a far more massive musculature to call into activity, than is the case with the arm-centres of the cortex. Again, the cortical centres for the upper extremities not only act through a greater range, but they innervate larger groups of muscles than do the centres for the head and neck, the muscles of articulation, deglutition, &c. It would, therefore, be natural to presume that the cortical cell-groups representing these respective regions would differ considerably in the size of their individual elements. The histology of the motor area fully warrants us in stating this to be the case; the smallest cells being found at the lower end of the central gyri and Broca's convolution—and thence increasing rapidly in size upwards towards the centre for the great musculature of the limbs, as illustrated by the following table of actual measurements:—

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* "The Brain of Mammals," *Syd. Soc. Trans.*

Comparative Size of Brain Cells.

	Average Size of Ganglion Cells.	Largest Cell—Maximum Size.
Left ascending frontal (upper end), .	60 μ × 25 μ	90 μ × 45 μ
Frontal gyri (areas at posterior end), .	45 μ × 20 μ	69 μ × 27 μ
Left ascending frontal (lower end), .	35 μ × 17 μ	41 μ × 18 μ
Left ascending parietal (upper end), .	66 μ × 41 μ	88 μ × 41 μ
,, ,, (middle third),	46 μ × 37 μ	55 μ × 32 μ
,, ,, (lower third), .	41 μ × 24 μ	...

We find this law fully borne out by the results of an examination of the bulbar and spinal cell-groups in different regions—the **greater musculature** being presided over by the groups of **largest cells.** We, therefore, see reason for regarding the dimensions of these cells in the cortex as influenced by—

(1) Range of discharging distance.
(2) Size of musculature innervated. (3) Age of nerve cell.
(4) Resulting multiplicity of cell connections.

It will be seen from these conclusions that the **deepest** elements are not necessarily the **oldest,** for some of the lowest of a series are very small and very simple in their connections. The reason for this was shrewdly given by Dr. Ross from observations on the development of the motor cell-groups in the anterior cornu of the spinal cord.* His statement is to the effect that the younger cells are in close contiguity to the blood-vessels: that as growth proceeds, they are thrust further aside, so that the larger and older cells lie midway between parallel vessels. No one familiar with the structure and disposition of the cortical elements of the brain will fail to see the force of this suggestion. These small pyramidal elements which we meet with constantly side by side with the older cells, are found often with very few lateral branchings, and the apex-process thins out rapidly and is lost to view at a short distance from the cell, notably contrasting in this respect with the older elements, whose apex-process can be traced up into the first layer or peripheral zone. It is important to note this fact—new elements are being continually formed, which for some time have *no connection with the grey meshwork* of the outer zone of the cortex. These extensions from the apex or centric pole of these young cells continue to thrust themselves further outwards, and are brought into apposition by delicate lateral offshoots, with nerve-fibre plexuses around.

Can we suggest the significance borne by the nucleus in the autonomy of the nerve cell? The results of physiological experimentation by Ferrier, Hitzig, Horsley, and Beevor, and clinical investigations, especially those of Hughlings-Jackson, appear conclusively to indicate the anterior or fronto-parietal realms of the cerebrum as especially motor; and the occipital and temporo-sphenoidal lobes as especially sensory, in their endowments; and it is, to say the least, highly suggestive that the large pyramidal and ganglionic cells peculiarly characterise the former, just as the smaller elements and densely aggregated granule cells characterise the latter—that, in fact, as we pass from motor to sensory realms, so we find the nerve cells progressively diminishing in bulk and the granule cell progressively preponderating in number. Dr. Hughlings-Jackson long since suggested the representation of *small* muscles by small cells, requiring as they would, in their almost ceaseless lively activity, rapid and frequent, though short, discharges of energy; in fact, he regards such small elements as necessarily of unstable equilibrium. His words are as follows:—

> "I have suggested that the size and shape of cells, as well as their nearness to the tumour, or other source of irritation, will have to do with their becoming unstable; other things equal, the same quantity of matter in many small cells will present a vastly greater surface to the contact of nutrient material than the same quantity in one large cell. I have also suggested that small muscles, or, more properly, movements which require little energy for the displacements they have to effect (those of the face and of the hands in touch, for example), are represented by small cells. Such movements are rapidly changing during many of the

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* *Diseases of the Nervous System*, vol. ii., p. 26, 1881.

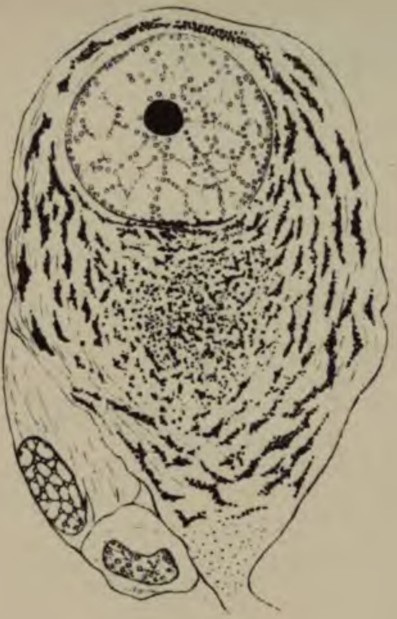
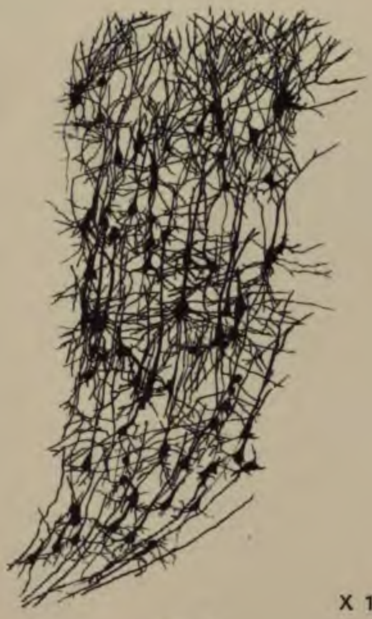


FIG. 1.



X 110.

FIG. 2.

CORTEX OF PIG'S BRAIN, TWO DAYS OLD,
SUBLIMATE PREPARATION.

Bale, Sons & Danielsson, Ltd., Lith.

operations they serve in—writing, for example—and require repetitions of short liberations of energy, and necessitate quick recuperation of the cells concerned. Movements of the upper arm are, in comparison, little changing, and require persistent steady liberation of energy." *

When, however, we consider the *assumed* sensory element of the cortex—the minute angular and granular cells—we must not lose sight of a remarkable distinction between them and the assumed motor unit, and that is, the great proportionate preponderance of the nucleus to the cell itself in the former. That the nucleus does exert some mysterious influence over the **nutritive** and **functional activity** of the cell has long been surmised; and the results of our histological inquiry indicate that nuclear degeneration within the nerve cell is peculiarly associated with certain states of mental and motorial instability. We have long been accustomed to regard it as related more definitely to the **functional activity** of the cell, and less *directly* related to the **nutritive activity** of the cell. In other words, the cell is subject to a constant supply of nutritive plasma—it gradually assumes a state of nutritive instability, and will necessarily discharge its accumulated energy in accordance with the simple law of **nutritive rhythm**—the resulting stable equilibrium is succeeded by a measurable period ere the potential energising of the cell has once more brought it up to its former state of instability. Were this all that occurs, the process of **storage** and **liberation of energy** would be a simpler rhythmic process than the more compounded rhythm which actually pertains to mental operations.

If, however, we regard the nucleus as affecting the functional activity of the cell, as in fact, restraining or **inhibiting its discharge,** as a kind of *imperium in imperio* exercising a controlling influence upon the perturbations which reach the cell from sensory surfaces: then the presence of a healthy nucleus would become an all-important feature in the cell-life—a feature of the utmost significance to us in our pathological enquiries. What really does occur when these nuclei are especially affected by morbid processes, we shall refer to more particularly in our chapter on the epileptic neuroses. The view we have here taken of the significance of the nucleus would lead to the conclusion that when, from its degeneration or morbid state, it fails to inhibit the cell, these nerve elements would be subject to a rapid *running-down* on trivial excitation, and in servile obedience to the law of nutritional rhythm; in fact, we should here find an explanation of morbid instability such as, *e.g.*, in motor realms results in convulsive states, and in the substrata of mental operations in varied psychical states and reductions in consciousness.

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* "On Temporary Paralysis after Epileptiform and Epileptic Seizures," *Brain*, vol. iii., footnote to p. 436.

It is these considerations which induce us to regard the disproportionately large nucleus of these small angular elements of the second layer of the cortex as being of some significance. Subject as such minute cells are to a rapid accumulation of energy, we might presume that some restraint must be established to prevent their reckless liberation of energy, and, hence, we believe such restraining capacity to be afforded by the very large nucleus. In the next place, we have every reason for believing that this superficial belt of angular cells is in direct functional connection with the subjacent cells of large size, and that their morbid instability would, therefore, affect these larger units, which, from the small size of their nucleus, would be more subject to the law of nutritional rhythm in their discharge of energy. As indicated by Dr. Ross, and also in the preceding note by Dr. Hughlings-Jackson, the large cell would present a far smaller area in contact with nutrient material than the same amount of protoplasm broken up into numerous minute elements; and hence, such large cells would labour under nutritive disadvantages—would be reservoirs for the *slow* accumulation and storage of energy, which, when liberated, would again result in a tardy re-instatement of nutritive instability.

Electrical Excitability of the Cortex.—Fritsch and Hitzig were the first to demonstrate, in the year 1870, the excitability of the cortex in animals to the galvanic current; and three years later Prof. Ferrier prosecuted with the faradaic current his first investigations into the functions of the cerebral hemispheres. The method of stimulation employed by Ferrier was, to use his own description, "The application of the electrodes of the secondary spiral of Du Bois-Reymond's induction coil, connected with a cell of the mean electro-motive power of one Daniell. The resistance in the primary coil was such as to give a maximum current of 1·9 absolute unit, as estimated for me by my colleague, Professor Adams. The induced current generated in the secondary coil at 8 cm. distance from the primary spiral was of a strength sufficient to cause a pungent, but quite bearable, sensation when the electrodes were placed on the tip of the tongue."* We can but briefly summarise here some of the more important facts elicited by these experimental methods respecting the reaction of the cortex to electric stimuli.

Latent Period of Stimulation and Summation of Stimuli.—It is from these phenomena we infer that the cortical areas found to be excitable are *really centres*, in the proper acceptation of the term. It must be remembered that a ganglionic centrum is an **elaborative structure,** and that stimuli applied to it meet with *delay* ere the resulting response be elicited. The excitation of a centre is therefore accompanied by the **time element** seen in nerve stimulation in a marked degree, and this is very appreciable in the stimulation of the so-called **psycho-motor centres** of the cortex. This is well brought out on contrasting the effects of a carefully-regulated current applied to the cortex of this realm, with the effects of the same current as applied to the medullated strands immediately beneath, by first excising the overlying cortex. In the first place, we find (after, of course, abstracting the time required for transmission down spinal cord and motor nerves and the latent period of the muscle) that the retardation is 0·045 of a second, and in the latter place, 0·03 of a second (*Franck* and *Pitres*). So, also, if very feeble stimuli be applied to the cortical centres their **summation** occurs, so that no contraction takes place until **several stimuli** have been delivered. Of the many interesting facts revealed by the researches of Schäfer and Horsley, Franck and Pitres, relative to the effect of electric stimuli on motor centres, the more important may be stated as follows:—

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* *Functions of the Brain*, 2nd edition, p. 223.

(1) In the *same* animal the **number of stimuli** per second requisite to produce a continuous contraction is always the same for cortex, motor nerve, and muscle.

(2) A **continuous contraction** does not occur on stimulating a motor centre, until the rate of stimuli reaches 46 per second; below this, single contraction occurs for each shock or thereabouts.

(3) The **contractile rhythm of muscle,** whether it be cortex, corona radiata, or spinal cord that is stimulated, has been shown to follow this rule:—

Rhythm of stimulus below 10 per second = muscular rhythm identical.
Rhythm of stimulus at above 10 per second = muscular rhythm constant and independent (*Schäfer and Horsley*).
Rhythm of stimulus about 46 per second = continuous muscular contraction (*Franck and Pitres*).

(4) The **muscular curve** of cortical stimulation is less sudden in its rise and more sustained than the curve shown in subcortical stimulation, and all voluntary muscular contractions show a similar rate of undulation in the muscular curve.

Modifying Circumstances.—The excitability of the cortical areas is subject to great variation. Thus, *different* animals vary in the intensity of stimulus required to produce the adaptive movement; and the *same* animal will vary from time to time as regards this susceptibility, according to the conditions in which it is placed. **Severe hæmorrhage** greatly reduces or even abolishes, whilst **moderate loss of blood** exalts, the excitability of these parts (*Munk*, *Hitzig*). Prolonged **exposure and stimulation** rapidly exhaust, whilst **apnœa,** and the **deep narcosis** of chloroform, ether, chloral, and morphia abolish it (*Schiff*), so that all animals completely anæsthetised fail to reveal such excitability. So if the cortex be in a state of **inflammatory irritation,** its excitability can be readily aroused by even mechanical stimuli, which in health have no such effect. In new-born puppies, Soltmann obtained early response to stimulus, of the corona radiata, whilst it was not until the *tenth day* that he was able to obtain such response by stimulation of the motor cortex.

Functional Equivalence.—Some authorities have inclined to the belief that a process of **functional compensation** occurs when injury, disease, or experiment has removed a motor centre: that either the opposite sound hemisphere, or even some other portion of the same hemisphere, may assume the functions of the area destroyed. It is undoubtedly true that centres bilaterally associated and least independent recover soonest from a lesion of one centre, and are least affected in the issue, as is indicated in the history of all cases of ordinary hemiplegia; but this can scarcely explain what we meet with in experiments on dogs. Here it has been shown that if the motor centres of one hemisphere be destroyed, the resulting hemiplegia is soon recovered from, and if this were due to the substitutional activity of the other hemisphere, ablation of the centres in the latter would presumably paralyse both sides. This, however, is not the case; for, as Carville and Duret clearly proved, the reinstated power of the limb first paralysed is not affected by the second operation. The explanation is, therefore, not one of functional substitution by another region, but is really due to the more **automatic character** of the movements in these animals; in other words, these movements are far more dependent upon the activity of lower centres and are less represented in psycho-motor or cortical realms. In man and the monkey such movements are brought more under the control of the volitional centres—they are removed, as it were, to a higher plane of activity, are less automatic, more independent, and their removal by disease or injury is followed by absolute paralysis of the opposite members.

Phenomena of Electric Stimulation of Cortex.—Professor Ferrier gives preference to the **faradic stimulation** of the cortex, rather than the galvanic, since the first requisite is a stimulus of a certain **duration,** and not the momentary effect of the opening and closing of a galvanic circuit; the latter also has the further objection of inducing electrolytic decomposition of the brain-surface if its action be long sustained. If the *intensity* of current be greater than necessary, **diffused stimulation** occurs, so that neighbouring areas are aroused into consentaneous activity.

Extra polar conduction has also been proved to occur by Carville and Duret, as seen in contractions of a frog's gastrocnemius, the sciatic nerve of which rested on the occiput of a brain, the motor area of which was stimulated. This fact, however, does not vitiate the results of a minimum current applied to the motor cortex.

Conduction to lower centres, as the basal ganglia, has been by some assumed to be explanatory of the results of stimulation of this motor area. This argument is, however, wholly disposed of by the fact that (1) *direct* stimulation of these ganglia (corpora striata) results in entirely different movements, not the adaptive, purposive movements which the psycho-motor centres elicit; and that (2) when we bring the electrodes upon their immediate superficial aspect, at the insula, no response whatever occurs. As might have been surmised, the radiations of the coronal medulla, entering into connection with the motor cortex, are in like manner functionally differentiated; and, as shown by Burdon-Sanderson, when the cortex is removed and they are stimulated, similar purposive movements can be called forth.

Proximity of Psycho-Motor Centres.—It has been seen that the so-called motor cortex, distinguished by the **nested cell-groups** of the fourth layer, is so distributed as to occupy distinct areas, separated only by narrow intervals from each other. This fully accords with the fact that the phenomena of electric stimulation of the cortex demonstrate the **close proximity** of wholly distinct centres, as Professor Ferrier remarks—"Areas in close proximity to each other, separated by a few millimetres or less, react to the electric current in a totally different manner." *

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* *Loc. cit.*, p. 229.

PART II.—CLINICAL SECTION.

General Contents.—States of Depression—States of Exaltation—Fulminating Psychoses—States of Mental Enfeeblement—Recurrent Insanity—Epileptic Insanity—General Paralysis of the Insane—Alcoholic Insanity—Insanity at the Periods of Puberty and Adolescence—At the Puerperal Period—At the Climacteric Epoch—Senile Insanity.

STATES OF DEPRESSION.

Contents.—Mental Depression Defined—Decline of Object-Consciousness—Rise of Subject-Consciousness—Muscular Element of Thought—Failure in the Relational Element of Mind—Sense of Environmental Resistance—Reductions to Automatic Levels—Sense of Effort—Restricted Volition—Enfeebled Representativeness—Transformations of Identity—The Physiological Aspect—Defective Circulation—Nutritional Impairment—Explosive Neuroses—Hunger of the Brain-Cell—Painful and Pleasurable Mental States—Reaction-Time in Melancholia—Degrees of Mental Depression—Clinical Varieties of Melancholia—Simple Melancholia—Delusional Melancholia—Hypochondriacal Melancholia—Melancholia Agitans—States of Mental Stupor—Stupor and Hypnotism—Acute Dementia.

PAINFUL mental states are of course *normal* under certain conditions in health and sanity. As in the intellectual sphere it is but human to err, so in the emotional sphere it is but human to suffer, and to feel acutely: hence it is not the *intensity* of mental pain (although this is often far greater than in healthy states similarly aroused) that characterises this phase of disease, for if the anguish be the outcome of commensurately painful circumstances, we regard it as but a natural reaction. It is in the fact that the emotional storm is out of all proportion to any exciting cause, that we recognise the departure from the standard of health. It is essential, therefore, that we carefully inquire into the antecedent circumstances of our patient's disorder, so as to determine whether there are adequate causes to account for the distress apparent—if so, there is but normal physiological reaction, and cerebral function cannot be regarded as deranged. If, however, the mental pain is the result of trivial exciting agencies, if moral or physical agencies arouse emotional states out of all proportion to what would occur in the healthy mind, then we infer that the grey cortex of the brain is so far disordered as to functionate abnormally, and we speak of the result as pathological depression. It is clear, therefore, that our chief difficulty in distinguishing normal from abnormal states of depression depends on our correct estimate of the correspondence of emotional states and their excitants, due allowance being made for special peculiarities of temperament. We cannot apply the same rule to a callous, unemotional nature as to one refined and sensitive.

In our search for adequate causes we do not confine our attention to the patient's environment; we must look for possible moral agencies, such as shock, disappointment, domestic affliction, together with physical agencies, such as injury, disease, privation, or, again, overstrain of mind, or vicious habits of life—in all alike, the real causes are centric, and consist in a disordered function—the incapacity of reacting commensurately in the conditions in which the organism is placed—in physiological terms it is a "disproportionately excessive" reaction. "The melancholia which precedes insanity is distinguished from the mental pain experienced by healthy persons by its excessive degree, by its more than ordinary protraction, by its becoming more and more independent of external influence, and by the other accessory affections which accompany it" (*Griesinger*).* By one thoughtful writer it has been suggested that melancholia might be spoken of as a homologous, while mania and monomania might be termed heterologous affections.† This, of course, would imply a quantitative and qualitative distinction; but, since emotional and intellectual states may be disordered qualitatively as well as quantitatively, the parallel is scarcely applicable. Emotional disturbances as the result of disease differ from the normal reactions of health, not only in volume but also in nature: as Herbert Spencer indicates, the correspondence may vary in two directions, quantitatively and qualitatively, in *degree* as well as in *kind*.

With respect to the non-relational feelings—the appetites, pains, &c.—Herbert Spencer says:—"Their great indefiniteness of limitation and accompanying want of cohesion forbid unions of them, either simultaneous or successive. Obviously, the emotions are characterised by a like want of combining power. A confused and changing chaos is produced by any of them which coexist."‡ This very want of relativity, this dissociability and absence of a tendency to form strong coherent groups, at once account for the comparative difficulty of estimating the degree of mental alienation in melancholia, as contrasted with states of delusion, where we are dealing with definitely measurable factors.

Simple pathological depression is ushered in by that **failure in object-consciousness** which invariably inaugurates a corresponding **rise in subject-consciousness**; and which, we have reason to infer, implies a diminished functional activity in those realms of the cerebrum correlated thereto. The patient exhibits a growing indifference to his former pursuits and pleasures: the ordinary duties of life and business become irksome and devoid of interest: especially do all forms of mental exertion cause ennui and distaste—the attention cannot as formerly be directed without undue effort, and so reading becomes laborious and thought sluggish and monotonous. The environment fails to call up pleasurable associations—a dreariness and gloom pervade the outside world, since it is interpreted in terms of the predominant feeling. All aspects of object-consciousness alike indicate the **negative state.** There is a want of vigour in the representation of the environment, and feelings aroused thereby are at a low ebb.

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* *Mental Diseases*, p. 210.

† *Psychological Medicine*, Bucknill & Tuke, 3rd edit., p. 440.

Principles of Psychology, vol. i., p. 177.

Corresponding to this there is a **rise in subject-consciousness,** shown in the prevalence of painful mental states—the predominance of gloomy emotions. This is the **positive aspect** of the patient's mental state, and this aspect is the one which chiefly obtrudes itself upon our notice. It is characterised especially by an all-prevailing gloom, varying in degree from mild depression up to acutely painful mental states. The subject may complain of vague anxiety—a feeling of some impending evil—an indefinite prevision of coming sorrow, which gives its own colouring to objective existences: he retires from social converse, which but adds to his irritation and mental distress, gives himself up to introspective states, in which he dwells upon the present contents of his mind, broods over his morbid feelings, and falls into long reveries, the subject-matter of which partakes of the same gloomy colouring. He is hyper-sensitive over trifles, irritable and impatient, or his querulous humour may alternate with sullen silence and obstinacy. Even in this reticence and retirement from social responsibilities, this growing apathy to all around or feeling amounting to dislike or direct hostility, we recognise the origin of that subjectivity, that egoistic state which, in more advanced affections of the mind, conjures up delusions of encroachment and persecution.

In every case of mental depression we have this duplex state to study—the negative affection of object-consciousness, and the positive affection of subject-consciousness.

Griesinger also asserts that forms of mental depression are due to states of cerebral *irritation* and mental *excitation;* but he apparently fails to recognise the duplex nature of the phenomena in neglecting the distinction between the two realms which comprise the totality of consciousness. Thus he says:—

> "In employing the term, 'states of mental depression,' we do not wish to be understood as implying that the nature of these states or conditions consists in inaction and weakness, or in the *suppression* of the mental or cerebral phenomena which accompany them. We have much more cause to assume that very violent *states of irritation* of the brain and excitation in the mental processes are here very often the cause; but the general result of these (mental and cerebral) processes is depression, or a painful state of mind. It is sufficient to recall the analogy to physical pain; and to those who imagine they make things better by substituting 'cerebral torpor' and 'cerebral irritation' for 'depression' and 'exaltation,'

> it may fairly enough be objected that in melancholia there is also a state of irritation." *

Had he asserted that both conditions co-existed, a state of cerebral **torpor** in the physical substrata of **object-consciousness,** and a state of cerebral **irritation** in the substrata of **subject-consciousness,** he would, we think, have faithfully recorded the morbid phenomena. The *normal* variations in these antithetic halves of consciousness, with which reverie and dreamy states render us familiar, have been thus lucidly expressed by Herbert Spencer, when in reference to the vivid and faint aggregates of consciousness he says :—

> "Though entire unconsciousness of things around us is rarely if ever reached, yet the consciousness of them may become very imperfect; and this imperfect consciousness, observe, results from the independence of the faint series becoming for the time so marked that very little of it clings to the vivid series." †

Decline in Object-consciousness.—The various states of consciousness and the changes from one to the other constitute collectively the sole elements of mind; and our considerations, therefore, apply to feelings and the relations between feelings. First, let us note that the variations from the normal state embrace a quantitative and a qualitative change. Feelings may succeed each other in *rapid order*, or in *slow, monotonous file;* they may arise in *serial order*, or numbers of disconnected states may simultaneously thrust themselves into the field of consciousness, producing turmoil and indefinite vague emotion and thought. On the other hand, mental phenomena may exhibit a qualitative alteration, such as, *e.g.*, is shown in degrees of intensity of feeling, or again, of definiteness as due to the more or less relational character of the product. The decline in object-consciousness which occurs in states of pathological depression, such as we are now dealing with, presents us with the following features :—

(*a*) Enfeebled representativeness : (*b*) a lessened seriality of thought (weakened attention) : (*c*) diminution or failure in the muscular element of thought.

The last appears to us so important a factor in these morbid states, as to demand here somewhat careful and detailed consideration.

Failure in the Muscular Element of Thought.—The constant accompaniment of depressed mental states is a diminished range of perception; and, since every perception is a complex phenomenon of composite states of consciousness—if one or other of the essential elements of an idea or of a presented object be wanting—the definite realisation of such object or idea is defective. The loss may be in the more sensuous element of the perception—in those qualities, in fact, of body which are categorised as **dynamic** ("primordial"), *e.g.*, colour, odour, taste, or the pure sensations appreciated by the specialised senses of sight, hearing, taste, or smell; again, the loss may pertain chiefly to the **statical** or **primary attributes** of the perception—those of size, position, form. A *vigorous perception of these* **primary** *or* **space attributes** *of body is dependent largely upon our "sixth" or* **muscular sense.** If, therefore, this sense undergo any diminution, so will the space attributes of body become less vividly conceived—*the cognition is but feebly produced.* The sense of sight is pre-eminently interwoven with the muscular mechanism involved in our perception of objects: and, since the retinal field can only receive the impress of these dynamic attributes of body by means of a musculature, which rotates the eyeball and so disposes the visual axis suitably, the knowledge of such movements, comprising figure, bulk, and position in space, becomes inextricably blended with these dynamic attributes.

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* "Mental Diseases," *Syd. Soc.*, p. 210.

† *Principles of Psychology*, vol. ii., p. 459.

There is little doubt that the *retinal impressions* are, in states of melancholic depression, but feebly produced; but whether the *muscular element* of perception is first or simultaneously affected, is an enquiry of special interest. And here we must distinguish between that portion of the muscular element which enters into our higher intellectual concepts, and that grosser factor of the large musculature of the limbs, &c., which subserves the purpose of locomotion and coarse movements. The sense of muscular contractions which forms the basis of the primordial ideas of form, size, position, lapses eventually in consciousness as a pure sense of muscular contraction. With the larger musculature this is not so: it is essential that the movements of the limbs, their contraction, and tension should be exquisitely registered centrally, as thereby alone can we gain an idea of their position in space apart from the sense of sight, and appreciate the relative weight of objects and the resistance offered by them. The unrestrained action of these muscles signalises to our minds the absence of external resistance, and the rise in the muscular sense which accompanies any resistance opposed is the direct measure of such resistance. Similarly, with the "**muscularity of thought,**" which in the normal state is of free and easy play, the rise into consciousness of its primordial muscular element means effort, and at once suggests to the mind the same notion of **resistance** in the **environment.** It is obvious, we think, that the muscular element is the first to decline: for cases of intense grief, as from a sudden mental shock, are associated with a notable contraction of this sphere, and space dimensions are altered and contracted. This feature is one of importance, since it clearly points to the decline of the more **relational elements** of the **perceptive process.**

The relations of bulk, configuration, and position are recognisable only by the intellectual operations of the mind, and it is this intellectual element which is earliest enfeebled. This follows, therefore, the inverse order of the evolution of psychical powers. Muscular sense, which appears much later in the evolution of the nervous system than do the general or the specialised sensations of sight, hearing, &c., is in morbid states the first to succumb. The infant learns to appreciate the colour of an object long before he has received the visual perception of its form, bulk, and position: he learns to recognise sounds ere the direction whence they proceed establishes the organised series of reflected changes in certain nuclei of the medulla, which enables him to turn the head and localise the source of such sounds.

Just as in the infant we trace the sensuous element of mind as preceding in evolution the relational element, so, in dissolutions of the nervous system in the insane, the inverse order is followed, and the relational decline before the sensuous or "primordial" sensations: and, since a relation can best be defined as a state of consciousness "*holding together* other states of consciousness" (*Herbert Spencer*), so individual conscious states become dissociated or unrelated. The loss of such relational element implies a certain degree of intellectual torpor; but, as we shall have reason to see, the sense of volitional freedom, which is probably an abstract product of the muscular sense, must in like manner decline. Our vigorous perception of the outside world depends largely upon vivid states of consciousness: our realisation of such related states by muscular sense and its derivatives may be compared to a **mental grasp** of the **environment:** and, in direct proportion to the vigour of such grasp, does our power over the environment predominate, and the **resistance** of the latter diminish. In states attended by decline of the muscular or relation element of mind, therefore, external resistance must be *pari passu* intensified, and the apparent *energy and freedom of the will restricted.*

Let us analyse this component of ideation more thoroughly, and we shall find that not only is every perception evolved from a series of complex related states of consciousness, but that every concrete perception or idea is attended by certain *vivid* primary states of consciousness and other secondary component impressions which fail to rise into consciousness, or are more or less revivable or representative. Now such unconscious components of an idea which we take, so to speak, for granted—these **lapsed states** of consciousness, although they form an integral component of the perception or ideal representation, are *chiefly of* **muscular origin.** If, in every conception of a sphere, the roll of the eyeball on its axis were induced, the objective origin of the perception of its form would be evident: but, although such actual muscular movements do not occur, yet the musculatures productive of such movements have their centres innervated by each such perception. Still, such innervation as a direct muscular state or sense of muscular tension and movement fails, in health, to rise into consciousness—an automatic play calls up vivid representation of form and figure without any consciousness of muscular action or strain. As before stated, the frequent repetitions of the muscular act essential to the knowledge of figure, position, &c., have eventually resulted in a lapse of the same muscular action in consciousness.

If, however, *delay* occur in the production of such relational states, the statical attributes of body will be perceived only after **conscious effort**; even actual muscular movement and the tension so brought about for the realisation of more vivid conception of form, configuration, and bulk, will give that sense of strange effort which metes out to us the *resistance of the environment.* Do actual muscular movements occur in the deranged states with which we are now concerned, and does **conscious effort** thus arise upon planes which are normally devoid of such feelings? The melancholic exhibits to a notable degree the effort which it causes him to think, reflect, or attend to what is said, or to what he reads. It appears to us that the true explanation is due to mental operations being reduced in level so far as to establish conscious effort in lieu of the usual unconscious operations, or lapsed states of consciousness which characterise all intellectual processes. The restless movements of the intellectual eye (in the artist, poet, &c.), as well as those of the state of maniacal excitement, bespeak in the former case the exalted muscular element of thought, and in the latter a highly reflex excitability; but in the melancholic these muscles of relational life are usually at rest, the eye is fixed, dull, heavy, sluggish in its movements and painful in effort, the eyelids are drooped, the limbs motionless. The only muscles in a state of tension are those which subserve emotional, and not relational life, viz., the small muscles of expression.

Hence, the failing vigour of representative states aroused in simple perception or ideation, issues in the sentiment of objective resistance. The environment encroaches *pari passu* with the failure of that faculty whereby the mind projects out of itself, so to speak, an environment, or revives in idea impressions received from the environment. It is the **motor element** of mind which is here at fault—the relational element of thought, since it is the **space attributes** of bodies which are involved. Now, since in the appreciation of these attributes of body (form, bulk, &c.) the subject is active and the object passive, it results that the motor constructive element of the idea is the one which suffers. In other words, failure in the muscular element of thought has as its results on the subjective side, enfeebled ideation and the sense of objective resistance.

With respect to the sense of resistance from the environment, it is of interest to note its artificial production in the reductions of consciousness by the agency of anæsthetics. To any one who has been anæsthetised, and who recalls his experiences, say, with nitrous oxide, it must be obvious how the environment crowds in upon one more and more, and how the ego, or personality enslaven by its power, finally feels that thought itself is succumbing to its resistless advance.

Restricted Volition.—As in the sphere of perception, so when taking into account consciousness in its totality, we likewise find the same failure in those complex muscular centres, which, in their adjustment to the environment, issue in what we term conduct. All volitional acts categorised under this head are the resultants of many factors, or rather the result of the struggle between many contending forces. A certain line of conduct or a certain action being determined upon, presupposes the representation in consciousness of the several possible lines of action. This, in other words, is equivalent to saying, that various feeble motor excitations are represented in consciousness, and that the stronger the aggregate of excitations in any special direction, the more does it tend to issue in action. Volitional actions are hence preceded by **nascent motor excitations.** Such excitations are the basis of the act represented to the mind **in ideas** which more or less vividly precede the act as **realised.**

In this conflict volition may be enfeebled as the result of failure of those initiative emotions, desires, and sentiments which are in abeyance in states of depression; or it may be convulsively restricted as the result of two opposing antagonistic forces, as when such groups of motor excitations divide the attention between them, and the mind sways from one to the other in hesitation and doubt; or, again, such motor excitation as forms the impulse to action cannot be definitely and strongly represented, and this enfeeblement of muscular representativeness issues in apathy and inaction.

A clearer conception of the resultant phenomena may be gleaned by contrasting the voluntary and the involuntary or automatic acts. In the latter, the ideal movements have lapsed in consciousness—the stimulus, whatever it be, is followed so rapidly by the appropriate reaction that the nascent motor excitations do *not* rise into consciousness. The start of surprise, the suddenly assumed attitude of self-defence, the mechanical movements employed in conveying food to the mouth, and the masticatory actions following thereupon, as well as other complex though automatic acts, have no initial motor antecedent represented in our consciousness; yet all these movements are exquisitely co-ordinated and rapidly executed.

In the enfeeblement of motor representations preceding volitional acts during states of depression, the actions themselves, if performed, are sluggish, mechanical, and devoid of normal energy; and herein lies the distinction between healthy automatism and these abnormal states. The distinction is more important than at first sight may be apparent, for upon it hinges the explanation of the **automatic**

freedom of maniacal states, which implies, as we shall see further on, a grave and more serious reduction. The apathy and sluggish reaction of melancholia appear in part due to this want of *vigorous motor representation;* the true characteristic of a normal and *vigorous mind* is the **vivid realisation** in consciousness of the action or line of conduct to be pursued—the ideal recognition of all alternative lines of conduct by the contrasting faculty, together with the representations of similar actions previously performed, with the result as affecting the organism.

In normal states, each group of the feelings which we class as desires and sentiments rapidly tends to swell the aggregate of its own motor excitations: so rapidly does this natural attraction of "like to like" go on, that the contrasting faculty whereby the result is obtained appears often to act with incalculable rapidity by a process which Spencer calls **"automatic segregation."** This process is impaired in states of depression, and becomes sluggish, feeble, and hesitating. Those faint summations of *ideal* movements which are aroused as the incitants to volitional acts may mutually antagonise each other; and their very want of vigour will of itself neutralise that distinctive quality which enables the one group to preponderate and overcome the other in action.

To employ a figurative illustration—thus do we witness in the surging tide advancing upon a rocky shore, two waves diverging at an angle; the one, receiving fresh impulses from minor wavelets which take the same course, swells into a rising crest; the other, receiving no additions, subsides exhausted. Or, two such waves of different size advancing the one upon the other, the higher, representing the aggregate swing of numerous undulations, overcomes and carries with it the surging elements of the weaker. Or, again, to illustrate the feeble representations alluded to, let us picture the uniform ripplets advancing by thousands on the surface; from want of co-operation, each maintains its own distance from the other, no great contrasting aggregate of movement is formed collecting to itself stray pulses of force, and hence all alike come to the shore with similar insignificant results.

Want of vigorous representation, enfeebled contrasting faculty of thought, antagonistic tendencies, or, lastly, recession or restriction of those feelings which normally excite to voluntary reactions may one or all take part in that restriction of the ego which we speak of as a restrained volition.

Here, again, we have suggested to the mind that *resistance* of the environment which inevitably results where subject-consciousness has a diminished range. It may at first sight appear contradictory to speak of a *fall* in object-consciousness and a *rise* in subject-consciousness as issuing in a sense of *resistance* from the object-world, and a state of enfeebled subjectivity: this is, however, the case, since the less definitely the mind conceives of external realities, the less vivid their representations—the wider the margin for doubt, suspicion, and ideas of encroachment from without. We fail to *grasp* the environment: we do not *know* it, in the sense of measuring our strength against it—*and hence we fear it.*

So again, the enfeeblement of subject-consciousness pertains only to that "faint aggregate of conscious states which the vivid aggregate tends to draw after them into being" (*Spencer*), viz., the ideas connected with the outside world, and the representation of our reactions upon the same—hence the faculties of ideation and volition are impaired. Far otherwise is it, however, with the more sentient element of the self-consciousness—that mass of bodily sensations, visceral, muscular, articular, cutaneous, and the feelings and emotions and sentiments which in the aggregate constitute the **sentient** or **passive ego**—it assumes a concentrated and exaggerated intensity, and this is what we refer to as the rise of subject-consciousness as distinguished from the decline of object-consciousness: a truly **self-analytic** state.

Failure of Personal Identity.—If, now, we attempt to trace further the decadence of mind, in progressive forms of mental disease, we arrive at a very notable stage, and one of profound import, when the failure of object-consciousness is so far advanced as to lead to alterations in the patient's notions of his relationship to the outer world, and to a confusion in his own identity.* A considerable difference is observable in these cases of confused identity, but the more important distinction appears to exist between—

(*a*) Cases of transformed identity associated with general feelings of regard or good-will to the outer world, and a universal sense of well-being, or, at all events, a complete indifference to the environment; and—

(*b*) Cases where, with the transformation of the ego, the environment or *non-ego* is also transformed in the patient's mind into a formidable, encroaching, and persecuting foe; whilst all its manifestations usually tend to call up a sense of repugnance and hostility. The ego may exist as a **double personality,** each independent of the other, or the one swayed by the other, and utterly dissentient in their nature. We need not here deal with these minor differences, but rather consider the development of the latter class, where the identity is transformed and the non-ego is estimated in terms of the malign. It is well, perhaps, at once to state, that these latter forms appear to us to arise out of the various *melancholic* types of alienation, whilst the former are educts of the more purely *maniacal* affections.

How does this mysterious transformation arise? The ego is constituted by the vast aggregate of sensations derived immediately from the body, which are a complexus of all grades of sensory manifestation, from visual and other special senses to tactual and general sense, as well as the far less definite organic or visceral sensations.

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* See on this point especially Ribot, p. 107-110; also Griesinger's *Mental Diseases*, p. 51; *cf.* Spencer, Sully.

All those ingoing currents which arouse, more or less definitely, our knowledge of the existence of a body, its limbs, musculature, and viscera, conjointly aid in the elaboration of the ego or personal identity. But the ego is far more than this. We must associate therewith those representations of the same, and moreover the "*faint aggregate*," as Spencer terms it, of states aroused by presentative cognitions of the outer kosmos.

Our sentiments, ideas, emotions, as well as our memory of presentative states, all alike go to form that complex elaboration—personal identity, which is severed sharply from the "vivid aggregate" known as the non-ego—the physical in contradistinction to the physiological environment. Now, since in all normal states, the internal order bears a definite relationship to the "outer order" of things, when either of these is profoundly disturbed, the identity tends to suffer considerably, as indicated by Sully. We are all acquainted with transient confusion of identity, in those waking states when we fail to realise the impressions suddenly received from the environment; and were the latter completely and *suddenly* transformed, we should fail to restore immediately the balance necessary to re-establish our own identity.

So, when the internal mechanism is deranged, and the orderly relationship of inner to outer kosmos is confused, personal identity is apt correspondingly to suffer. We have already seen how this may occur in the progressive failure of object consciousness.

The failure to appreciate external relationships, again, is associated with that gathering gloom, that sense of outward resistance, fear, and insecurity of the non-ego already alluded to. Impressions from the outer world fail to arouse the normal representative states of cognition, but aid in the welling-up of the *emotional life* of the subject, and it is from this latter source that **falsifications of sense** arise.

As subject-consciousness becomes more and more pronounced with failure of object-consciousness, all impressions alike, received from the non-ego, become the pabulum for the growth of an all-pervading **egoism.** The subject broods over his multiform and novel feelings—morbid introspection and egoistic musings replace the healthy altruistic feelings and sentiments: and, since the emotional life is itself in part the origin of representative cognitions of the outer kosmos, *so out of this source there now arise falsifications of the environment.*

The pervading gloom, the sense of objective restriction, and the emotional states so aroused, attract to themselves like groupings of ideas—"**attempts at explanation,**" as Griesinger has it; and this state progressing, tends eventually to the establishment of a **new nexus of ideas** correlated to impressions received from without, in lieu of the old and normal relationships pre-existing. It would be a fallacy to assume that the falsifications of the environment *precede* the emotional disturbance, or that delusions of persecution beget gloomy and malignant passions—this would really invert the actual sequence of phenomena. A gloomy emotional background begets a gloomy interpretation of the non-ego, and all delusions of persecution are begot in like manner out of disordered emotional states.

Such translations, if we may so speak, from emotional realms to the realms of thought are, even in normal states of mental life, of frequent occurrence: they peculiarly characterise the poetic faculty, and distinguish the purely emotional and imaginative from the intellectual type of mind; but, where such emotional incitants to thought are in themselves the product of morbid action, the intellectual result of such operations is liable to be delusive and false. The more *immediate* concepts, as we may term those which are the result of pure intellectual operations, unassisted by, or only associated with, emotional states, are more subordinate to accurate laws of logic: the more *mediate* concepts, emotionally derived, are less susceptible to such exactitude of classifying and grouping. Such concepts, in the morbid states now under consideration, are utterly illogical, unclassifiable, fragmentary, and betray but the *disjecta membra* of a once rational mind. It appears to us that such distinctions between the immediate and the mediate knowledge, so acquired in the case of the insane, are all-important in our conception of the genesis of these morbid conditions of the ego.

We have been tracing in these mental operations the transformation of the environment to the alien's mind: out of the old tissue, by a species of re-arrangement and reconstruction, is woven a fabric representing to him the reality of external things, and which to him is the *only* reality, but, to his former state of sanity, is an utter falsification. Since this morbid concept is projected out as the actual kosmos, and since internal order must correspond to the external, so a transformation of the ego itself responds to this altered state—the former identity is lost and replaced by the new.

And here we have an explication of that **newly-acquired freedom** which, at this juncture, appears to dawn upon the mind of the monomaniac. No longer are phenomena in the outer world laboriously investigated and subordinated to rigid laws of logic and of science—they pass, as through a magic crucible, the morbid tissue of his brain, and are transformed in accordance with no objective laws, but take their colour wholly from the morbid emotional states present. Self-creations arise with wondrous celerity and of protean form; and the morbid imagination conjures up fantastic groupings utterly devoid of coherence and objective reality. A feeling of new freedom replaces the old one of restriction and aggression by the environment, and the ego is consequently endowed with new faculties, new powers—becomes a mighty potentate or a god. Still, the environment is indelibly stamped with the malign character which the former emotional state fostered, and it is only in late stages of the malady that such realisation of a new-got freedom entirely effaces the enmity of the non-ego from the mind.

Like all sudden and extensive transformations of mind, the change thus delineated must be accompanied, as Griesinger has indicated, by great emotional disturbance, "as the results of the conflict between the old and the new." He says, referring to the new sensations and instincts which become generated :—

> "At first these stand opposed to the old *I* in the character of a foreign *thou*, often exciting amazement and fear. Frequently their forcible entrance into the whole sphere of the perception is felt as if it were the possession of the old I by an obscure and irresistible power, and the fact of such forcible possession is expressed by fantastic images. But this duplicity, this conflict of the old I against the new inadequate groups of ideas, is always accompanied by painful opposing sensations, by emotional states, and by violent emotions." *

It will be seen that we differ from the above statement, in regarding the emotional perturbation *not* as the outcome of the "conflict between the old and the new ego," since it appears more in accord with the sequence of the phenomena to regard the morbid emotional storm of this period as being the direct origin of the newly-generated identity.

Reductions such as ensue from nervous dissolutions *alone*, can scarcely explain the phenomena with which we meet: we must, in addition, suppose a process of **re-integration** to ensue.

The level to which the mental life is reduced is still one of active, nascent, mental life,† and, like all such nascent life, is accompanied by much emotional disturbance. Even in these morbid minds there is no reason to suppose that the same process does not proceed which we assume to occur in profound sleep, where the *re-energising* in lower planes, while the individual is for the time unconscious, still proceeds, and so mental potentialities are unconsciously acquired. So also in the monomaniac, though the activities be those of lower planes, still they indicate **developmental activities,** and those groups of sensations and ideas are conserved which are the fittest to survive : irrational as may be the beliefs, inconsistent the new concepts with the actual truth, still, as Hughlings-Jackson indicates, they are the best possible in the patient's state of reduction.‡

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* "Mental Diseases," *Syd. Soc.*, p. 50.

† Dr. Hughlings-Jackson has repeatedly insisted upon the negative and positive results of epileptic seizures.

‡ We are prone, by the loose phraseology of common life, to regard the subjective as a permanent possession—to speak of our mind as a something beyond the simple active contents of the moment and as the accumulated psychical activity of our total existence; as if thoughts could be bottled-up permanently and unchangeably. It is the material substratum of thought—the organised nervous plexuses—which represents the permanent and the potential revivabilities of former experiences, as Herbert Spencer says :—"Just as the external *nexus* is that which continues to exist amid transitory appearances, so the internal *nexus* is that which continues to exist amid transitory ideas."—*Principles of Psychology*, p. 485.

As the tide of *intellectual life* retires, so does it well-up into emotional states; but such emotional wave must have its rebound, and this is expressed in the re-integration which pervades the mental organism with fresh ideas and concepts; and when such groups acquire a certain **definite cohesion** amongst themselves, we have the **genesis of a new identity.**

It is only at an advanced stage of dissolution that this transformation of the ego can occur—we may safely assert that extensive connections between distant nervous mechanisms must be deranged or dissolved, ere that failure of association of ideas could occur which always precedes this morbid change. Fresh connections probably arise, through the newly-forced channels of the emotional wave, and new centres of internal cohesion are begot and evolve the fresh association of ideas of the transformed ego.

And here we might note what we shall later on deal with more fully—viz., those transformations of the personality which characterise certain critical or climacteric periods of life—notably that of puberty. It can readily be conceived how powerfully the mental life is affected by the re-integration of the new encroaching sensations into fresh instincts, desires, impulses; or, as at the menopause, by the ablation, so to speak, of one of the strongest instincts of the nervous constitution, the sexual. Can it be a matter of wonder that, at these critical periods, the risk to the mental integrity should be great or that, in many subjects, permanent damage should ensue? So interwoven are these instincts with the whole fabric of mind, that a complete transformation of the sentiments and feelings follows, as the result of such incorporation. Obscure longings and yearnings, imperfect, indefinite perceptions, emotional surgings which have no obvious origin or purpose, characterise a period of perturbation of the mental life, which may readily lead to misdirected efforts or morbid impulse and disease.*

The Physiological Aspect.—In dealing with states of mental depression, did we attempt anything like an artificial division of this class of the vesaniæ, it would appear to us more important to lay emphasis upon the morbid processes to which they are traced, whenever such processes can with justice be assumed. It is clear that the symptomatic indications of the so-called varieties of melancholia point not so much to a fundamental distinction in their essential nature as to one in their mode of origin: they indicate quantitative as well as qualitative variations in the nutritive functions of the nervous centres, and, hence, are roughly divisible into groups, comprising those which arise from direct disturbance of the blood-current, and those which are induced in the nervous tissues primarily.

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* See on this subject the section on the "Insanities of the Period of Pubescence."

Two groups stand strongly contrasted here: the one, in which a **defective cerebral circulation** is the *more prominent* feature; the other, in which an acute **nutritional anomaly** of the nerve-centres expresses itself in still more unmistakable symptoms. A further group may be constituted by the various qualitative variations of the blood-plasma—**toxæmia,** &c.—a group conveniently placed between the two former.

D'Abundo has recently affirmed that the toxic and bactericidal action of the defibrinated blood-serum is much increased in all forms of insanity, except in mental depression, in which, on the contrary, it is lessened. His experiments demonstrated the fact that some 10 c.c. of serum to the kilogramme of blood kills rabbits by acute intoxication. It by no means follows, however, that mental symptoms due to the products of auto-intoxication should bear any direct relationship to the toxic effect on lower animals of the blood-serum injected. The effect of certain poisons administered to rabbits upon the nerve cell and its histological constituents has recently been carefully studied by Nissl, very definite changes being induced in the nerve cells of the spinal cord and brain by such agencies as alcohol, morphia, strychnine, lead, phosphorus, and arsenic.*

It will at once be evident that this is a very arbitrary grouping, the one condition being often associated with the other—nay, evolved out of the other. Thus, defective circulation leads eventually to grave nutritional anomalies, so that the symptoms of the first group may pass into those of the third, although the usual result is not its passage into the *acute* but into *chronic* forms of nutritional impairment. Again, quantitative and qualitative variations of the blood, affecting centric nutrition, may co-exist, whilst such nutritional disturbance of the nerve-centres reacts again upon their blood supply. Yet this inter-dependence of functionally related systems, although it renders any sharp demarcation into separate groups impossible, does not impair the practical value of a division into the three groups, since it always holds good that we may clearly distinguish those affections in which the *prominent indication* is that of simple *depressed circulation,* from a state in which the *vitiated quality* of the blood chiefly appeals to us, and, lastly, from those grave affections in which *acute* and *chronic nutritional anomalies* are the chief factors concerned.

States of defective circulation will comprise all the simpler forms of melancholia characterised by lowered cerebral activity. Excitations from the environment do not arouse the normal reaction; they are sluggishly transmitted, slowly elaborated, and wholly fail to react with due vigour or purposive result. The registry of all impressions is faint or imperfect, the latent period prolonged, the reaction-time delayed.

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* *Rivista Sper. di Freniatria*, vol. xviii.

The very earliest signs preceding genuine pathological depression are really the symptoms of cerebral anæmia and nervous exhaustion. The cerebral functions are torpid, there is diminished activity both of the impressive and of the expressive realms of the cortex, as above described, and negative states predominate throughout. The subject is heavy, languid, sleepy; frequent yawning occurs—not the *insomnia* of a more advanced stage; intellectual efforts are oppressive, and thought becomes dreary, monotonous, and painful. If the warnings thus afforded be disregarded, there arises the *frequent* **recurrence** of a **painful idea,** *occasional* **sensory hallucination, sleeplessness,** all indicative of a commencing pathological change—of impaired centric nutrition. In the earlier stage, where warning is not taken, and where, despite such clear evidence of cerebral exhaustion, the brain is still made to do its daily round of duty, in a state utterly inadequate for such exertion, unless absolute rest be here enjoined, the next step will certainly issue in **pathological depression.** The morbid nature of this change is sufficiently evident in the fact, that the diurnal cycles of nutritional rhythm are frequently inverted, or at least gravely disturbed.

Viewed from the mental aspect, the highest psychical operations are first enfeebled: abstract thought becomes oppressive or impossible; attention impaired or restricted; sensations are less vivid, and perception is incomplete or wanting in detail or imaginative vigour—the representative faculty especially being enfeebled. Apathy and indifference to the surroundings, associated with painful gloom, pervade the mind, betraying the decline of object-consciousness and the rise of subject-consciousness.[*] In these states, the reaction on the outer world may be characterised by fitful irritability, impatient conduct, sluggish, mechanical actions, or by entire suppression of volitional initiative. Both sensorial and motorial functions are sluggish or in abeyance, and the functions of organic life are all depressed. The vitality of the organism as a whole, being largely dependent upon the activity of the nervous centres, must necessarily suffer when this important regulative system is deranged: the condition is truly one of **devitalisation**—life is carried on **at a lower level.**

Should the nutrition of the nerve-centres suffer *materially*, a fresh series of symptoms is aroused: illusory states and hallucinations distract the attention—the mental pain and disquiet is intensified thereby: apathy and indifference may be replaced by timidity, fright, or terror; and the reaction becomes expressive of such emotional states—restless movement and agitated demeanour replacing the former negative condition. All this indicates impaired nutrition of the nerve-centres, owing to the defective supply of blood: the nerve cells, impoverished, exchange their normal functional irritability for an exaggerated **abnormal explosiveness,** and fitful irregular discharges replace the rhythmic outflow of the nervous discharges which regulate the subordinate centres and relational apparatus of animal life.

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[*] So the converse of an over-active circulation reveals itself in increased cerebral activity—often in extraordinarily vivid memories. This we see in fevers, also after the use of certain drugs, as opium, hashish, &c.—(Ribot, *Op. cit.*, p. 198).

These nutritional anomalies reach their climax in the third group to which I have referred, the **explosive** or **fulminating psychoses.** These affections are characterised by the suddenness and explosive nature of the nervous discharge, which relieves the pent-up and accumulating energy of highly unstable centres. In lieu of the equable rhythm of discharge and repair, corresponding to the wants of the organism, and adapting it to its environment, there is disproportionate accumulation of energy; the centres are brought up to a degree of high nutritional instability, and the least excitant, however trivial, may, like the spark to the fulminate, issue in an explosion of serious intensity. The **nerve pulse** is irregular, fitful, intermittent.

This group comprises certain varieties of so-called **impulsive insanity**—the homicidal and suicidal—the subjects of epileptic neuroses, and affections arising at the climacteric cycles.

The cortical expanse of the cerebral hemispheres is certainly the site of the highly representative and re-representative operations: a defective circulation here results in a genuine **starvation** of the **nerve-elements.** How does this starvation betray itself? In replying to this enquiry let us briefly refer to the physiological appetites for food, &c., and parallelise them with the case in point: we shall then find that all animal appetites are dependent upon two essential factors:—

(*a*) The reception of peripheral excitations by a centric register.

(*b*) The supply of blood to this centre.

Thus, the sense of hunger is the indication of a want of this due excitation of the peripheral nerves of the gastric mucous membrane; and for its alleviation the centres must receive impressions so created. But excitation of the peripheral ends of the vagus, produced by any mechanical contact other than by the ingestion of aliment, temporarily suffices to restore the nutritive equilibrium of the nerve-centres. The rhythmic pulse of excitations thus transmitted to the centrum calls up the increased vascular flux associated with all brain functionising—and thus, these two agencies combine to raise the nerve-elements into their normal physiological condition of satiety. The reinstatement of molecular equilibrium in the centric nerve cells depends not alone upon the transmission of the physiological stimuli, but also upon the *collateral flow of blood to the part.* So—as regards the special senses—the abolition of the usual afferent impressions begets a condition which is truly a pathological hunger. Strikingly is this the case with the sense of *hearing:* depression of spirits is a well-marked phenomenon in suddenly-induced deafness, partial or complete. The depression so induced we regard as a genuine instance of sensorial hunger—as the expression of starvation of the nerve cells, thus deprived of the normal ingoing currents.

Sameness and monotony of sensory impressions produce identical states: and the want of "a change" is nothing more than the expression of this physiological hunger of the nerve-centres.

On the other hand, the more highly representative the special sense faculty is in its evolution—the *less dependent* is it in this respect upon presentative excitations; and thus the sense of *sight*, when similarly affected, fails to indicate in the same notable degree a corresponding depression of the emotions—idealising or centric initiation so completely supplementing the loss that the results are far different: yet, if the sphere of such operations is in itself implicated, if the nervous mechanisms initiating representative processes are starved out by deficiency of blood, then there is begotten a corresponding hunger of the brain-cell. For, cerebral activity in these realms being restricted, as shown in the poverty of active ideation and thought, there is an arrest of diffusion-currents provocative of the pleasurable emotional states which always accompany healthy energising of these centres. Corresponding, therefore, to the dreariness of thought in cognitive realms, we have in the region of feeling such **painful mental depression** as accords with what we should term the **hunger of the brain cell.**

Again, those ganglionic structures which are the regulative centres for the organs of vegetative life, subserve the wants of the system through the agency of an inscrutable law of nutritional rhythm, differing for each organ concerned: yet, whether we consider the ganglia connected with the visceral sensations, or those which receive epi-peripheral excitations, as those of the special senses—*i.e.*, whether the physiological stimuli are continuously received, or have intervals of some duration, or, as in the case of the heart, are equable and periodic—in all cases alike, the excitation of such centres depends much in its degree upon a due supply of blood to the part; unless this be the case, the centre, exhausted by discharge and not renovated by due nutritional flux, must lose in its excitability.

Further, this exhaustion means a *weakening of that associative affinity* which arouses correlative centres, and which is the physical basis of ideal association. In like manner, the directive agency of such exhausted centres must be enfeebled, and the blending of impressions and associated states into the "serial line of thought" (*Spencer*) must be correspondingly enfeebled. As we shall see further on, this weaving of the crude material into forms of thought becomes a greater and yet greater *effort*: **"ganglionic friction,"** as Romanes aptly terms it, becomes arrestive of the higher processes of thought, and this resistance in the *intellectual* sphere is associated with a diffusion towards the more purely *emotional* sphere.

Painful and Pleasurable Mental States.—Since these states form so important an element in conditions of mental depression and exaltation, it will repay us here to summarise briefly our views as to their nature.

Mental pain has been defined as the result of *under-action* or *over-action*—its antithesis, pleasure, finding a place midway between these extremes; as though, we might say figuratively, an ocean of sluggish waters and of stormy billows lay on each side respectively, with a mid-region of rippling sun-lit wavelets. We think this definition fails—under-action certainly leads to apathy and torpor—over-action to all the various grades of painful mental states; yet, the essence of this mental pain is surely not over-action, but **pent-up activity.** Mental pain varies in degree from mild indefinite gloom up to extremes of anguish and despair, in which restricted volition is replaced by agitated and frenzied movement. Now, immediately the sphere of *object-consciousness* declines in functional activity, the *minus* quantity of the one sphere becomes the *plus* quantity of the other; which, in physiological terms, implies that ingoing nervous currents which normally would arouse appropriate reactions in the intellectual and motor realms, become diffused in the realms of feeling and emotion: what is lost for perception is gained in feeling. The restricted accumulation of energy is surely at the basis of states of mental pain. If we allude to such states as the result of *under-action*, the under-action is distinctly that of the higher planes, whilst there is a corresponding *surplus of activity* aroused in the subordinate planes of feeling and emotion. The so-called states of *over-action*, again, are *similar conditions more definitely expressed*—the over-action being that of the recipient, afferent, or impressive sphere, with a corresponding under-action of the efferent, intellectual, and expressive sphere—in fact, all grades of mental pain are dependent upon **over-action** on the **impressive** and **restricted activity** on the **expressive plane.**

In *normal* states, ingoing currents, or impressions centrally initiated, are translated into realms of motor activity or high intellectual phases; in states of mental pain, such translation is restricted, and such activities expend their energy in those diffused spreading discharges which are the correlatives of emotional conditions.

We find *pleasurable* states invariably associated with the translation of *feeling* into *thought* and *action*: we likewise find *painful* mental states associated with the surging tide of feeling vainly struggling to burst the barriers, in order that it may appear under the varied forms of intellectual or muscular activities: yet, we find *all degrees* of the latter—from that of high-strung emotional potentiality, down to those minor states where feeling is expressed in terms of general gloom or irritable impatience and fretfulness—passing, in fact, towards states where feeling as a higher emotional state seems well-nigh abolished, and passive indifference and apathy indicate a purely *negative* state of mind. These latter cannot be comprised under the head of states of mental pain, however consistently they may be classed as states of mental *depression* or *anorexia*.

The Reaction-Time in Melancholia.—Any estimate of the reaction-time in health or disease must take account of many possible sources of error; and such fallacies are but intensified when dealing with the insane mind. A large proportion of the insane do not, of course, admit of such methods of examination: and even amongst such as cheerfully respond to experimentation, a certain proportion are likely to falsify results from individual peculiarities, and the unpredicable vagaries of the insane: delusional cases are in this connection the most doubtful subjects. Where the reductions involve much impairment of memory, or profound mental torpor, the test of reaction-time, however taken, is perfectly futile and unreliable: and it is only in those instances of *incipient mental derangement*, where the *intellectual operations* are not *grossly* involved, that a failure in the energy of cerebral reflex can be regarded as of important significance.

In applying the test, the patient should, so far as possible, be made to take an interest in the experiment, and this can frequently be done with great success, by a little tact, even in serious cases of melancholic depression, or acute maniacal outbursts. Each subject should be repeatedly tested short of actual fatigue; and no average struck of the rapidity of reaction from less than *twenty* trials. In our own experiments with the insane we have restricted ourselves to the estimation of the total time required for reaction to the stimulus of sound or light; and have not attempted to investigate the more complex reaction-time of a more involved process.

Reaction-Time Instrument.—We have employed an instrument made by the Cambridge Scientific Instrument Company and designed by Mr. Galton, and have found it admirably adapted for our purpose in testing the reactions in the insane. A short description of this apparatus (fig. 18) may not be out of place here.*

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* See description of this apparatus by the author in Hack Tuke's *Dictionary of Psychological Medicine*, vol. ii., Art. "Psycho-physical Methods," No. 3, p. 1022.

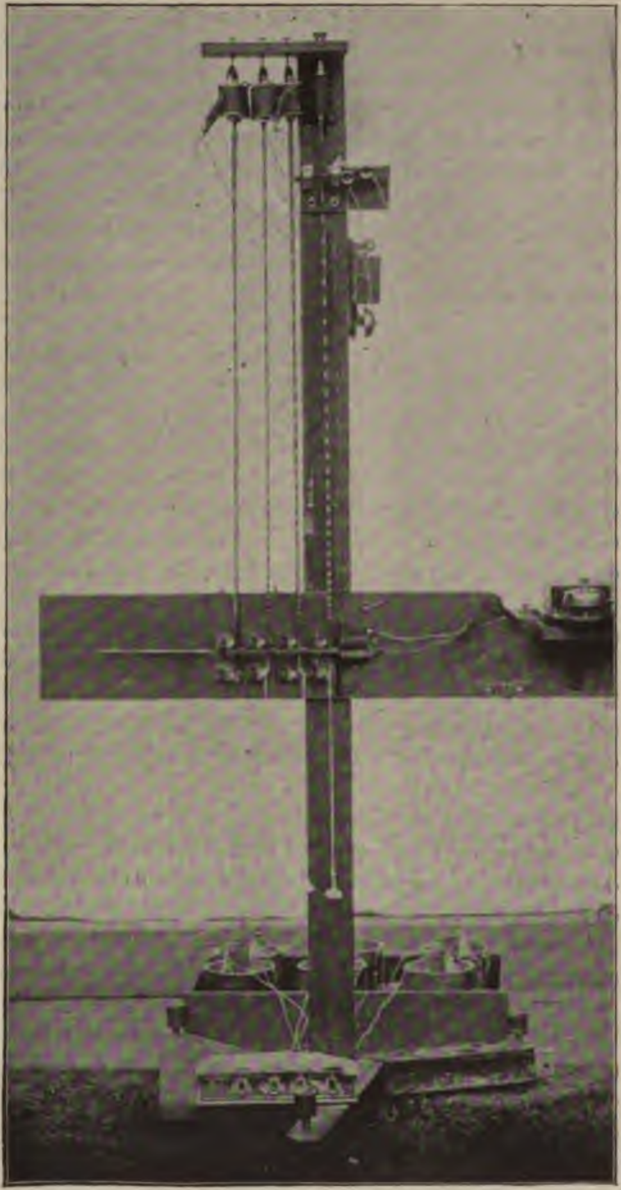

Fig. 18.—Reaction-Time Apparatus.

The above represents the apparatus as subsequently modified for obtaining a *prolonged* reaction-time. It will be noted that four graduated rods take the place of a single rod; that each rod is suspended by an electro-magnet to secure silent release, and that the six Bunsen cells (quart) supply the current for working the electric signals, the upper series of magnets, and the electro-magnet for the clamp. The simpler form of apparatus—devoid of the extra rods, battery, and foot-board—was the instrument mainly used for the results given throughout this work.

A square standard of pitch pine, 5 feet 10 inches in height, is fixed into a solid, unyielding tripod, in which levelling screws ensure its exactly vertical position. Half way down this standard a rectangular piece of mahogany or teak is screwed at right angles to its long axis; this supports a horizontal table upon which rests the hand of the party to be tested. To the same rectangular piece a small electro-magnet is fixed, which holds in position (as an armature) a spring stirrup so long as the electric circuit remains unbroken. This latter circuit passes over the table to a contact breaker, so that the finger of the operator, by depressing a button here, breaks the circuit, releases the stirrup, which, being in its turn drawn back by a powerful spiral spring, clamps the registry rod in its fall. The steel base of the stirrup is fitted inside with rubber, thus forming a more effective brake.

From the summit of the standard a box- or lancewood rod is suspended, three feet in length, and accurately graduated along its edge in hundredths of a second, up to thirty divisions, the limit of its complete registry being therefore three-tenths of a second. Astride the summit of the registry rod rides a heavy brass plate which falls a short distance with the rod, being then arrested by a diaphragm, its impact causing the **sound** signal; or by the make and break of an electric current here it starts an electric bell as the sound signal. The registry rod as it hangs suspended is concealed from the subject's view by a narrow projecting ledge of pine wood fitted to the standard betwixt the rod and the person to be tested. In this ledge, at a convenient height for the eye, is a small vertical slit or window, and a corresponding slit exists in the rod through which the light is seen; but, on the release of the rod, the window is closed by its fall, and a **sight** signal is thus afforded.

This apparatus should be as simple as possible, solid and absolutely steady; the rod should hang perfectly vertical and should not come in contact with any surface in its fall; by arranging the levelling screws in the tripod the clamping of the rod should secure it from any sliding, and in its descent the rebound should be reduced to a minimum at the foot, and as little clatter as possible allowed. The release of the rod should be effected with absolute silence, and this is best secured by suspending it to a straight bar electro-magnet by a short cylinder of soft iron. The fall of the rod should be on a cushion or pad filled with sand to deaden the resulting thud and check the rebound. The rod is released by pressing a button conveniently placed behind the standard out of observation, which breaks the circuit of the electro-magnet suspending the rod.

The Test.—The subject sits supporting his right hand on the table, his forefinger on the interrupting button of the clamp-magnet. The operator silently releases the rod which gives the sound signal, and the

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forefinger is instantly depressed, releasing the stirrup and clamping the rod. The figure on the front of the rod where clamped gives the reaction-time for the **sound** signal.

In the next place the brass weight is removed and the subject, sitting as before, is directed to keep his eye on the light seen through the slit in the rod. The rod is now released, the light disappears, and the subject, as before, clamps the rod as rapidly as possible. The time taken in the fall, as read off on the edge of the graduated rod, gives the total reaction-time for a **sight** signal.

The general results, so far obtained, would indicate a *decided prolongation of the reaction-time* in many forms of insanity. Simple affective forms—as in melancholic depression or maniacal excitement of a simple nature—as well as insanity, the outcome of alcoholism, or of epilepsy, and associated with general paralysis—were made the subjects of enquiry. In none of these were the results more strikingly uniform than in *alcoholic forms* of insanity, where, after eliminating every probable source of fallacy, the reaction to an optic stimulus was almost invariably delayed, and, in most instances, the reaction to the acoustic stimulus was likewise involved. None of the patients tested suffered from any serious degree of dementia, such as would have prevented their fully entering into the interest of the trial.*

In *general paralysis*, also, the same delay in reaction occurred, but for such cases we must refer the reader to the series of experiments as given in the section treating of these forms of derangement. Here we more particularly desire to record the results obtained from the subjects labouring under melancholic depression, simple or otherwise. In the following table we have contrasted the results obtained from a series of individuals presumed to be healthy, and from the subjects of more or less acute melancholic depression:—

REACTION-TIME IN HEALTH AND DISEASE.

	Acoustic Stimulus. Sec.	Optic Stimulus. Sec.
Self,	·13	·16
R. H.,	·15	·17
T. H.,	·16	·18
R. L.,	·13	·21
D. A.,	·16	·21
R. W., *Simple Melancholia,*	·29	·30
M. L., ,, ,,	·22	·25
S. W., *Climacteric Melancholia,*	·29	·29
J. W., *Hypochondriacal* ,,	·23	·24
G. A., *Delusional* ,,	·20	·26
C. K., ,, ,,	·14	·24

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* See also "Reaction-time in certain Forms of Insanity," in Tuke's *Dictionary of Psychological Medicine*, vol. ii.; also "Reaction-time" (in same), by Prof. Jastrow, with Bibliography.

With the above we also contrast the results given in a table by von Kries and Auerbach, embracing the investigations of several observers *:—

Observer.	Acoustic Stimulus. Sec.	Optic Stimulus. Sec.
Hirsch,	0·149	0·200
Hankel,	0·151	0·225
Donders,	0·180	0·188
Von Wittich,	0·182	0·194
Wundt,	0·128	0·175
Exner,	0·136	0·150
Auerbach,	0·122	0·191
Von Kries,	0·120	0·193

It will be apparent from the observations on healthy subjects, that whereas from $\frac{12}{100}$ to $\frac{18}{100}$ of a second formed the limit of variability for *acoustic stimuli*, and $\frac{15}{100}$ to $\frac{22}{100}$ for *visual stimuli*—in the insane, the former is only exceptionally below $\frac{20}{100}$, and the latter rises from $\frac{24}{100}$ to $\frac{30}{100}$ of a second. In healthy states the reaction to visual stimuli is slower than to acoustic impressions:—

> There seems good reason to suppose that the reaction-time of sight is necessarily longer than that of hearing or touch, on account of the photo-chemical nature of its more immediate stimulus. One observer (von Wittich) has even gone so far as to conjecture that the speed of conduction in the optic nerve is less than that of the other nerves of sense; it is rather to be concluded, however, that the latent time of the sensory end-apparatus, and of the cerebral processes by which sensory impulses pass over into motor impulses is different (*Ladd*). †

The prolongation of the reaction-time in cases of insanity *generally*, would indicate a special impairment in the visual as contrasted with the auditory sphere: both are often involved; but the former often suffers to the exclusion of the latter, it being frequently observed that a subject who responds readily and normally to an acoustic stimulus, exhibits notable delay in the response to a visual stimulus.

There are many reasons for agreement with Professor Ladd that the distinction in reaction-time for these two kinds of stimuli is due, not to a different **rate of conduction**, but to the **different atent period of end- and centric-organs**: and we may assume, with nearly as much certainty, that in the deranged states met with in the insane, the protraction of reaction-time found is due, either to implication of the sensory end-organ, or to the intra-central link whereby the sensory is transformed into the motor impulse; the former is probably illustrated by certain subjects of chronic alcoholism ‡—the latter in ordinary forms of affective insanity.

Degrees of Mental Depression.—Of the innumerable combinations of mental symptoms embraced under states of mental depression, certain forms present themselves, having many features in common of sufficient distinctive value to constitute them arbitrary varieties for the purposes of systematic study: such so-called varieties, however, it must be understood, are by no means other than purely artificial or arbitrary divisions, which are nevertheless essential for the orderly grouping before the mind's eye of what otherwise would form but a chaotic and confusing assemblage of facts.

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* *Archiv. f. Anat. u. Physiol.*, *Physiolog. Abth.*, 1877.

† *Elements of Physiological Psychology*, p. 477.

‡ *Vide infra*, under *Alcoholic Insanity*.

It is thus we hear of a purely **affective melancholia,** in which the emotional or affective sphere is chiefly at fault; and of a **delusional melancholia,** in which the intellectual or ideational sphere suffers.

Whilst fully recognising the utility of such grouping—whereby we keep in view the more notable affective implication in the one case, and the more prominent intellectual perversion in the other—we must insist that the student is here likely to fall into the serious error of regarding such arbitrary divisions as the *negation* of a principle which we regard as one of the greatest importance in our studies of insanity—viz., the **universality of implication** which characterises mental disease.

By this universality of implication we do not mean that all mental faculties suffer alike in extent or degree—this would be obviously absurd: but, that however prominent and obtrusive may be the implication of any special faculty—however limited at first sight may appear the derangement, further investigation shows that the *mind in its totality* has suffered. The psychological aspect of mental depression presented to the student in the foregoing remarks, will have prepared him for the recognition of this fact of the universal implication of the mental sphere in cases of morbid depression—as *subject-consciousness* rises in intensity, so he has learnt to appreciate the wane of *object-consciousness.* It matters not how mild the form of pathological depression—how slight the degree of mental pain—object-consciousness invariably presents this corresponding enfeeblement: but this latter feature has to be carefully looked for, whilst the former is the obtrusive and prominent indication of the derangement.*

We do not here allude to *delusional* perversions, but simply to those minor grades of failing representativeness which we have already traced in the sluggishness and poverty of ideation, its lessened vigour, and the dubiety of mind respecting objective existences, which, later on, culminates in delusions of suspicion. When we consider how, in transient functional disturbances falling *far short* of *pathological* depression, we find a gloomy emotional tone associated so frequently with a morbid suspicion, bordering at times upon actual delusive states—inconsistent, irrational misjudgments of our fellow-men and universal distrust, we may be fully prepared in states of genuine melancholia, however mild in type, to recognise in the sphere of the intellectual operations a corresponding wane.

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* These considerations sufficiently indicate the fallacies of implication to the student's mind of the term *partial* as contrasted with *generalised insanity.*

Whilst *minor degrees* of pathological reduction result in a welling-up of feeling as the more obtrusive feature (simple melancholia)—*deeper reductions*, resulting in more serious implication of representative operations, issue in *delusive perversions* (delusional melancholia) as the more striking feature, whilst the emotional gloom, in its place, aids in the creation of further delusional notions as "**attempts at explanation**," to use Griesinger's phraseology.

It will at once be apparent how this view differs from that which enunciates, as its leading doctrine, an *affective* origin for insanity: our own view being that the *relational* and the *sentient* elements of mind must be conjointly implicated, and that the *priority* of implication pertains to the *relational.*

Of the clinical groups arbitrarily constituted, from amongst the subjects of mental depression, we may cite as the more important—

(*a*) Simple melancholia.
(*b*) Melancholia with delusions, including the hypochondriacal form.
(*c*) Melancholia agitans.
(*d*) Melancholia atonita, or melancholy with stupor.

To these separate groups we must now devote some attention: in the first place, it is necessary to indicate, that the varied states which these terms connote are the outcome of the same morbid process in the cerebral cortex, and represent but *different depths of dissolution—serial stages in the same disease.*

A still lower stage of reduction is that of maniacal excitement: and we mention this fact here, since it is so frequently implied that mania and melancholia are *distinct diseases*, rather than different stages of the same morbid process.

(*a*) **Simple Melancholia.**—Under the term of simple melancholia are embraced forms of a purely emotional or affective insanity, where there is mental pain or emotional distress apart from obvious intellectual disturbance—if such mental pain be abnormal in its intensity and disproportionate to any exciting cause, we have a species of simple melancholia.

Here, at the outset, we must qualify the phrase, "a purely emotional or affective insanity:" for it requires but little insight into the operations of the sound mind, to lead us to the conclusion that so interblended are all the mental faculties in their mutual co-operation, that no such division can be drawn, in a strictly scientific sense, between the purely emotional and the intellectual states. When we speak of emotional states, we must ever bear in mind that the term connotes more or less of the intellectual element of mind—that every mental operation presupposes in its very simplest form—feeling, memory, reason, volition; or rather that these are but different aspects of the same state. It is, therefore, only in the greater preponderance of the one or the other factor that we distinguish between abnormal mental states.

Simple melancholia, therefore, really embraces those states of morbid depression in which the painful emotional element of mind preponderates to the exclusion of disorder of the more relational element; or, to be more exact, where the disordered feelings by their intensity and obtrusiveness overshadow any slight intellectual disorder which may be present. Definite delusional states, therefore, are evidently excluded from our definition; reason still asserts herself; there is no enfeeblement of memory; volitional control is not withdrawn. The cerebral dissolutions which such states of melancholia imply tend certainly towards a lower level, towards more complete dissolutions—and the psychical expressions then vary with it to those of disordered reason, memory, and will: yet, for purposes of clinical study, it is convenient, although this tendency be obvious throughout the attack of insanity, to fix the mind's eye upon the *affective* disorder.

The subject may long have struggled against the gradually increasing depression, and may have concealed his actual state from the notice of relatives or associates—any undue reticence, absence of natural buoyancy, or change in demeanour being usually explained away upon any other grounds than those of *mental* implication: and thus the barrier between simple functional disturbance and a genuine pathological process is passed without notice. A universal gloom pervades his mind, and a distaste for all previous avocations and interests declares itself: exercise and all forms of recreation no longer appeal to him, and a dull uniform level of indifference is engendered towards the outside world. Life has lost its freshness—Nature presents him with no delights, and whatever there be of beauty or happiness or gaiety around, but serves to emphasise his gloom as he feels their want of kinship to his nature. With still greater emphasis can he say with Nature's poet:

> "But yet I know, where'er I go,
> That there hath pass'd away a glory from the earth."

Retiring into the solitude of his own self-consciousness, he broods abstractedly over his alien state—fully cognisant of the nature of his malady; often dreading to reveal his condition to those most interested in his welfare.

But though the object-world has lost for him its pleasurable aspects, and thought and feeling with regard thereto are laboured, restricted, and wanting in vigour—yet subjective states of introspection, of self-analytic activity are keenly dominant, and this self-inflicted torture grows apace as sleep is lost, as defective appetite and sedentary habits of life still further retard the processes of nutrition and repair, and sap the foundations of his mental vigour.

It is at this period that **suicidal promptings** often come to the front; but, here we see Reason asserting herself—the patient recognises his moral obligations clearly—often shrinks with horror from the suggestion—or may be driven to implore protection such as may be afforded by asylum supervision. Many, however, in the gentle forms of depression, are equally conscious of a degree of self-control which enables them to meet any such suggestion with perfect confidence: they may utter the usual formula of being wearied of life, but, with the utmost self-assurance, deny that they could ever be induced to lay violent hands upon themselves. As we shall see later on, it is in much more serious nutritional anomalies, that the helplessness of the victim and the dread of impulsive acts prevail, as in the fulminating psychoses. Every degree of mental pain may prevail in the subjects of simple melancholia, from such as do not materially interfere with their pursuits—home or business duties, to such as result in utter paralysis of volitional energy: and, in these cases, their daily wants have to be strictly attended to—as they would starve rather than exert themselves to eat.

It is by no means unusual at this stage of depression for homicidal actions to replace the suicidal deed. From motives of intense love for her offspring—to save them from the terrible calamity which appears to await herself—the mother will occasionally sacrifice her children or anyone nearest and dearest to her (*Nicholson*).*

If the patient does not improve, a further stage is reached in which we observe a still greater wane in *object-consciousness:* the jaundiced view of the environment is no longer correctly interpreted as due to the subject's own indisposition, but doubts arise—distrust prevails—and a **suspicious bearing** towards those around inaugurates this further state of dissolution. The patient, not actually deluded, begins to misinterpret all interference, however kindly meant—looks suspiciously at his nurse—struggles violently at the most trivial attention paid him; whilst the preparations for feeding him or other necessary procedures may be met with every sign of terror. And yet, on questioning him, he admits no definite deluded state—is readily reassured—only the next moment to relapse into his state of all-prevailing fear that something may happen—he knows not what. The volitional restriction here is serious—self-confidence is greatly enfeebled, and suicide is not unusual: the stage is one of transition to the more definitely deluded or acutely melancholic forms. The following is a typical illustration of simple melancholic depression:—

M. A. W., aged sixty-three, a married woman with a family of two children, was admitted suffering from an attack of depression, which had commenced about three weeks previously. She had an earthy complexion—the cheeks mottled with dilated capillaries: her bodily condition approached the obese. No pronounced cardio-vascular change was apparent, although the heart's action was somewhat feeble: the genito-urinary system appeared healthy. It was stated that she had just attempted to drown herself in a water-butt. For three weeks past she had slept but little; had become more depressed from day to day. From her friends' statement, it appeared that she had led a perfectly steady, temperate life; had never before exhibited any mental disturbance or eccentricity, and was not known to have had insane or neurotic ancestors. She was extremely depressed, wept constantly, was reluctant to enter into particulars about her mental state. She admitted that she had been failing in health for some months prior to her attack, and that she did not know the cause of her bodily or mental ailment—could not explain why she was distressed, but was constantly the subject of vague fear, and frequently asked what was to become of her. No delusions were apparent: she had suffered from no hallucinations. This condition continued for some three weeks: she always presented a most melancholic expression, but slept well at night without sedatives, and only on one occasion required forcible administration of food. Through the day she sat rocking herself to and fro, sobbing aloud, and at times became greatly agitated. The more acute symptoms then subsided, and she turned her attention to household work. She became more reticent, and when pressed with questions grew miserable and wept bitterly: could still give no explanation for her fretting. In less than a month she fully realised her own improvement in health—grew more hopeful, less reticent, but now troubled herself much at having attempted suicide. She was now given small doses of opium and ether (15 and 10 minims respectively) twice daily, and in a few weeks later was cheerful, active, industrious, longing to return to her friends and home, and left the asylum some ten weeks after her admission.

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* See Dr. Nicholson's remarks at the discussion of Dr. Semalaigne's Art. on the "Insanity of Persecution," *Journ. of Mental Science*, vol. xl., p. 516.

(*b*) **Delusional Melancholia.**—This form, as before stated, we regard as presenting us with a deeper stage of reduction than that of the simple form of affective insanity (simple melancholia). Gloomy apprehension and suspicion have here passed into definite and persistent delusional states; and, intense as may be the emotional implication, the intellectual derangement now appeals more forcibly to us: and, being constantly insisted upon by the patient, is apt to be regarded by the friends as the real *cause* of the malady. It would be quite apart from our purpose here, even if it were practicable, to illustrate the various features assumed by these cases of delusional melancholia—they will receive sufficient notice in the several clinical forms of insanity which we shall deal with later on. The perversions of the intellect may apply to any one of the whole range of things outside the subject, or may be entirely restricted to the bodily and organic sensations: or again, to his relationships to another state of existence—to his moral being. An infinity of delusive notions, therefore, necessarily presents itself, often in such strange and contrasting combinations as to be utterly unclassifiable.

Prominent, however, amongst such delusive notions are those which deal with the subject's corporeal frame—the head, the body, limbs, or viscera—often of a grim, and as often of a grotesque character, and which, if the attention be riveted thereupon, constitute the so-called **hypochondriacal melancholia.** Then again, we meet with delusions relative to the moral being—the victim has committed the unpardonable sin, or for some, perhaps, insignificant action, his soul is entirely lost—or passages in Scripture constantly recur to him of a gloomy denunciatory nature as applicable to his own state, forming one of the class of so-called **religious melancholia.** Or again, the encroachment of the environment is the more perceptible feature—and the mind conjures up those malign agencies therein which are expressed in the multitudinous **ideas of persecution,** tyranny, treachery. And yet again, a well-marked class of patients infer **demoniacal possession, witchcraft** or other unseen agency as accounting for their states of mental perturbation. In these conditions of delusional melancholia, hallucinations are not only frequent, but often form the chief material out of which such delusional states are framed. Aural hallucinations more frequently occur than visual, and both far more generally than affections of smell or taste. Hallucinations of smell are of ominous import—they are frequently associations of irreparable alcoholic brain disease—of epileptic states—of traumatic forms of brain disease, &c. In the following case, however, we find such hallucinations of smell in acute insanity induced by alcohol, but rapidly recovered from :—

M. A. S., a married woman, aged forty-eight, suffering from her first attack of insanity, stated to have been of ten days' duration. She had lived an immoral life for twelve years past, and lately had been of intemperate habits, drinking heavily up to the onset of her attack of insanity. She is not known to have inherited insanity. She was of corpulent proportions, her complexion dusky, and expression dissipated : her bodily health had not very materially suffered. On admission she was greatly agitated and terrified, shaking her limbs violently in bed, or trying to rush from the room. She slept for a few hours after taking 30 minims of paraldehyde. Much melancholic agitation continued next day : she struggled violently to open the doors, declaring "there was a charm to open them," that there was "a woman after her to burn her," that "she *saw* the flames." The following night six hours' sleep ensued upon the administration of 25 grains of chloral : she awoke calm and orderly.

At this period she was composed, attentive, and coherent, but betrayed the presence of numerous delusions and recent aural hallucinations : thought she was in an asylum : came here for protection from the noise in her own house. There was such shouting and calling ; *if she did not answer, they got louder and louder.* Voices kept calling, "Mrs. Birkett, Mrs. Smith, Mrs. Birkett, Mrs. Smith, come and help me, come and help me out !" when she asked, "Who has put you there ?" they shouted "Rustan, Rustan, Rustan ;" that had been the cry in her house for the last two years. (She had gone to the police for protection against a man she called Rustan, and had stated that she then saw his body blown up by dynamite.) She had "*smelt an earthy smell like that of a dead body* in her house for the past few weeks." When she went to the police for protection, "hundreds of blackguards followed shouting after her." She went the same day to get water from a tap in the yard. "She was sure it was drugged, it dried up her mouth, which began burning and swelling; she was confident somebody wanted to stab her." As usual in such cases, she absolutely repudiated any suggestion of intemperate habits.

Her calmness of demeanour continued, but she required a sedative each night to secure any sleep. A week later she affirmed that at home she constantly heard a voice from beneath a stone table calling out, "O Mrs. Rustan, Mrs. Rustan, O Mrs. Birkett, O Amy, come down here; I'm down here under the stone." She went and searched under the stone, and saw what "would have blown her up if she had remained in the house." None of these voices have been heard since coming to the asylum.

A fortnight after her admission she exhibited but very gentle depression, and for the first time began to question the real or imaginary nature of the voices heard. No relapse occurred, and in less than two months from admission she left, perfectly recovered.

(*c*) **Hypochondriacal Melancholia.**—In this form of delusional insanity, the morbid interest of the patient is concentrated upon his bodily organism and its functions. In healthy states of activity the ingoing currents arouse, as we have seen (p. 160), none but the massive feeling of *pleasurable well-being*, and it is only when the bodily functions are deranged that we become directly conscious of the existence of our organs. So interblended, so inextricably interwoven is the web of sensuous feeling produced by such activities, that out of it arises the *central core* of the personality—the *ego*: around the latter there crowd the impressions received from objective existences—the *physical* in contradistinction to the *physiological* environment; yet, these two halves are dissevered, and although they help to form the aggregate mind of the individual, the characteristic stamp of healthful mental operations consists in the continuous and vivid realisation of this distinction between the subject and the object-world.

In the lower forms of life, we conceive of the *subjective* element as forming by far the larger factor of mental states—a vast series of impressions received from the physical environment are *not* referred thereto; and, although the appropriate reaction may occur, this by no means proves that such sensations are not referred to some part of the organic or physiological environment. The higher we rise, the more definite becomes the reference of its own series of excitations to the object-world; and this, in certain special lines, we see to a remarkable degree in certain insects, such as bees and ants. In man, of course, we attain the complete severance between these antithetic halves which renders possible his knowledge of nature as embraced in the various sciences.

We have seen how the failure of the one half (object-consciousness) may proceed to a serious extent without implicating the groundwork of our being—the personality; and states of advanced *dementia* realise this still more fully: we may equally well conceive how the other half (subject-consciousness) may suffer disruption, if this "**sensuous core**" of the **personality** be implicated either by peripheral or centric derangements. That the former is possible, we have confirmed by numerous instances of hypochondriasis with mental derangement, arising from disease of the abdominal and thoracic viscera; that the latter occurs, is sufficiently obvious in the excitation of similar states by *mental* agencies—the perusal of morbid and sensational books, obscene pamphlets, and the association with similar cases of hypochondriasis.

The anxieties and delusions of the hypochondriacal patient may have reference to any part of his bodily organisation: amongst the insane, however, prominence is given to the tract innervated by the *pneumogastric* nerve, and thus the regions of the throat, thorax, and abdomen—the respiratory, circulatory, and gastro-intestinal organs—are peculiarly the subjects of the patient's anxious attention and complaint.

The hypochondriacal subjects of epileptic insanity almost invariably refer their ailments to the stomach and bowels—obscure feelings, pains or imaginary diseases, torpidity or obstruction, are incessantly dwelt upon by them; and in most instances, if not all, have some basis in actual derangement; but it is in the constant brooding over these states, and the exaggerated colouring of their ailments, that the hypochondriacal condition is revealed.

In the alcoholic subject, on the other hand, hypochondriacal notions have reference often to the peripheral ends of the nerves of common sensation: thus, they continuously examine their limbs, complain of pricklings and other strange sensations in the skin; assert that they are poisoned, so that the skin is black, diseased, or "corrupted" (see *Alcoholic Insanity*).

To take the more frequent ailments complained of in hypochondriacal melancholia, there is the idea so frequently leading to obstinate refusal of food, that "the throat is made up," or that the gullet is wanting, an idea which persists in spite of the frequent passage of the feeding-tube. In such cases, a spasmodic stricture of the œsophagus is not infrequently met with as an obstruction in feeding—the spasm is always high up: it is a reflex spastic state intensified at once by the introduction of the œsophageal tube. Although met with in men, it is of more frequent occurrence in women, and then often associated with functional uterine disturbances, as in the œsophagismus of hysterical subjects. Organic stricture we have very rarely met with in such subjects; but direct compression from enlarged thyroid we have frequently found, associated with such delusive conceptions of the absence or total occlusion of the gullet. Such patients are intensely dejected, often seen with the head bent forward, the hands grasping the throat, and fully persuaded that they are dying of inanition, whilst fed artificially with ample meals. They will point to their limbs (often well nourished) as evidence of their advanced emaciation; and they will often induce vomiting, by irritating the fauces after feeding, declaring that the food so introduced can do them no good.

In like manner, other subjects declare that they have no stomach, and transform various dyspeptic symptoms into indications of grave disease: they may on these grounds resent any attempt at feeding, and struggle violently to thwart one's efforts. Others may take food heartily, yet declare it does not nourish them, and that they are slowly undergoing starvation.

Obstruction of the bowels is a most frequent idea, aperient medicine is asked for repeatedly, and despite the daily action of the bowels, the insane patient reiterates his belief that no stool has been passed for days or weeks. Such patients are pictures of misery, importunate about their treatment, querulous, irritable, wholly absorbed in their own feelings, and can be induced to talk upon no subject without at once reverting to their miserable plight.

In other cases, the genital organs are the source of anxiety—the subject believes himself to be impotent or the subject of syphilis, and no possible argument can be used to assure him that his whole system is not permeated by the virus. One patient, at the West Riding Asylum, believed his generative organs had been displaced: another that his sexual organs were diseased and mortified. Numbness of the epigastrium was a sore grievance to another patient, who, moreover, believed that his stomach contained pins and needles. In the case of a middle-aged man who died of tubercular phthisis, an accident (from which he had really suffered some years since, and in which he fractured an arm and two ribs) was made the basis of extravagant delusional notions. He insisted that his skull was nearly hollow—"half his brains having been scattered about at the time;" that he also lost "two gallons of blood;" and that a screw placed in his bowels by some unknown agency caused him continual and terrible agony. The strangest combinations of delusional notions arise out of the most trivial disturbances of function—slight constipation, flatulence, heartburn, eructations, mild intestinal catarrh are exaggerated to ludicrous proportions: the subject has a huge animal within him gnawing at his vitals, or is full of serpents; or his meat and drink are poisoned by vitriol; he is "full up inside;" or flames of fire continually burn within him. A male patient who died at Wakefield Asylum of the marasmus induced by long continued refusal of food and melancholia, was wont to believe himself covered with a skin eruption from head to foot; he would also blow his nose forcibly to demonstrate how his brain was gradually passing out by that channel.

Patients, again, will lie in bed declaring their inability to rise because they have no body or no legs: and one well-known character at Wakefield, when asked her name, would always reply, "I have no name; I am no-one; I have no body, no head, no limbs; I'm a voice; I'm an echo."

Burrows speaks of hypochondriasis as never occurring before the age of twenty-five;* but *hypochondriacal melancholia* is by no means infrequent at puberty: in fact, we might well expect the onset of such a disturbance from what we know of the physiological cycle of events which occurs at this age (see *Insanity of Puberty*). "Hypochondriacal states are sometimes observed in the years of childhood, and more frequently at the age of puberty. They are extraordinarily frequent in young people, and more rare in advanced age" (*Griesinger*).†

We notice in all forms of hypochondriacal melancholia, one feature wholly distinct from that characterising most forms of simple mental depression, and that is the insatiable craving for sympathy in place of reticence and self-retirement. This tendency renders hypochondriacal subjects the most unpleasantly egoistic, and the most tedious of all cases of insanity: it induces them frequently to stoop to any depth of deception, and to ape almost any condition so as to attract attention. It is in such instances that the hypochondriac so closely approaches the hysterical type, that it becomes a moot point how to distinguish the one from the other. One of the most striking instances of the kind which I have met with, was that of a young tabetic subject, in whom cerebral disturbance supervened, and in whom this morbid craving for sympathy led to simulation of many symptoms, such as voluntarily induced eructations, retching, and exaggeration of his genuine tabetic state, and then to the most mendacious and vindictive, yet groundless attacks upon the attendants and medical officers.‡ In like manner, we find patients who will lie in bed, forcibly and continuously eructating, or obtrusively shamming efforts at vomiting to attract the attention of the passer-by. In a case to be referred to later on, the patient utters loud exclamations of distress as the medical officer approaches; or induces startings of his limbs, which he refers to electric shocks passing through his frame; or makes hideous grimaces, if he thinks he is observed, rolling his eyeballs about as if in torture. In another instance of hypochondriasis, a female endeavoured, for months together, to attract notice by loud belching noises, but found a more ready means of commanding attention by picking, scratching, and defacing her forehead and cheeks with her nails, presenting a most piteous aspect: scarcely had she recovered ere she recommenced the same practice, and only appeared satisfied by the sympathy it evoked. It is in this craving for sympathy and self-reference that genuine hypochondriacal insanity differs from the hypochondriacal stage of delusional insanity—*i.e.*, of the progressive systematised insanities: in the latter, as we shall see later on, the reference of the subject is invariably to the environment; the cause of all his misery is outside, and in lieu of the craving for sympathy, a hostile feeling is rapidly engendered.

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* *Commentaries*, p. 466.

† *Op. cit.*, p. 217.

‡ The case was one of interest throughout, and has been fully detailed by my colleague, Dr. F. St. John Bullen, *Brain*, part xli., April, 1888.

Occasionally, the morbid epigastric sensations induce unnatural cravings. as is the case with hysteric subjects: we have known the case of a female hypochondriac advanced in years, who, in this state, cleared away gradually a square yard or more of plaster from a wall by continuously swallowing small fragments, ere the cause of the disappearance of the plaster was detected. This same patient subsequently took to pulling out the hair of her head and swallowing it; by this means, she had become on two occasions completely bald over the scalp: ere she died, she manifested the still more revolting habit of devouring excrement. Yet, this woman even declared she had *no body*, and would moan piteously for hours at her forlorn condition.

Another aged hypochondriac would restlessly pace the rooms and corridors of the asylum day by day, bleating like a goat in distressful tones, a picture of abject misery. He had been fed for months together twice daily, but his evening meal, purposely concealed on a scullery shelf by the attendant who had discovered his weakness, he would always secure and drink surreptitiously; yet he could never be induced to take his other meals by a similar stratagem. In these cases of chronic hypochondriacal melancholia in advanced age, dementia progressively advances and no recovery is to be anticipated.

Suicidal tendencies are presumed by the friends to exist in all cases of hypochondriacal melancholia, and we frequently hear of attempts at strangling, hanging, drowning, or other measures in which we may fairly conclude that a *bona fide* suicide was not the patient's object, but rather a morbid wish to attract attention to his case: in fact, these subjects very rarely make such attempts within the walls of an asylum. Occasionally, however, as the outcome of alcoholic intemperance, we meet with a form of melancholic hypochondriasis, which, once recognised, will not again be readily overlooked: it is one of hypochondriacal delusions associated with extreme enfeeblement of the will, and desperate impulsive conduct is its invariable accompaniment (*J. F.*, *J. S.*). Such cases are highly neurotic by heritage.

(*d*) **Melancholia Agitans.**—We have referred to delusional forms of melancholia as a deeper reduction than the simple affective form: now such cases of delusional insanity frequently exhibit acute symptoms*—*i.e.*, restlessness, incessant movement, insufferable anguish, and every indication of an agonised state of mind—these forms of *acute* melancholia are still deeper stages in reduction: they are in every sense an approach to the maniacal reductions. By *melancholia agitans*, we do not indicate this acuteness or intensity of mental pain, for the actual pain is often far more superficial in character than might at first sight be apparent: but we denote by this term the prevalence of a **motor agitation,** which, in like manner, approximates to the **maniacal states.** Such forms of melancholia may be of short, but usually are of prolonged, duration, even lasting over several years; they may form but a stage in any mental disturbance, or may characterise the case throughout, to its termination in recovery, in dementia, or in death. Dr. Fleury recognises the intensity of suicidal proclivities, as well as the unfavourable prognosis of agitated melancholia occurring at the climacteric in women; he also draws attention to the frequent association of such disturbance with drinking propensities.*

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* Acute in the sense of intensity, not of duration.

The patient is quiet only when asleep; rocks her body to-and-fro, or paces up and down the room incessantly; the hands are in constant movement, grasping the head, tearing the hair, rubbing the chest, picking the skin until it bleeds, biting the nails, tearing or disarranging the clothing—or huddled in a corner, her face buried in her hands. she sways to-and-fro, lamenting her fate in loud sobbing or ejaculations sufficiently expressive of mental distress. Almost invariably the delusive ideas from which she suffers are prominent from the first—her soul is eternally lost; she is cast out from God and the world; she is disgraced, or has brought ruin upon herself and family.

Hallucinations do not appear to prevail in this form—they may have occurred as a prelude to this stage of reduction, and in their recall may constitute material for delusive ideas. The hypochondriacal forms which we have just studied may exhibit this state of motor agitation at different periods of its course. Let us take as our illustration of this form of mental depression the following case, where, after an onset of acute melancholic reductions and grave moral perversions, the patient passed into this chronic stage of melancholia agitans:—

S. A. A., aged 52, and married. The medical certificate runs as follows—"Will not leave her bed, rocks herself about, moaning and repeating that she is doomed to go to hell. Says she can see the flames of hell before her eyes: that it is of no use eating or doing anything in the house, as she is bound to go to the bad place." Her daughter states that patient got out of bed to strike her, and said: "I could tear you all to pieces: all my love is turned to hatred." Here, then, we have a case of acute melancholia, utterly unfitted for home treatment—tortured by hallucinations of the senses, and delusions based thereupon: also by impulses to violence—"I could tear you all to pieces"; and grave moral perversion, as indicated in her confession to her daughter—"All my love is turned to hatred."

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* "Clinical Notes on agitated Melancholia in Women," by Dr. E. L. Fleury, *Journ. of Mental Science*, July, 1895, p. 548.

These impulses, if neglected, would just as readily issue in a suicidal act. They indicate extreme instability of nerve-tissue, and the explosion must occur in some form of suicidal, homicidal, or generally destructive conduct. The patient had a similar attack of three months' duration, some twenty years ago, occurring after labour. She has had considerable anxiety caused her by a drunken husband, who has squandered all his means, and lost much property. *A brother of the patient hanged himself.*

The acuteness of the patient's symptoms rapidly subsided after admission, but she still remains the subject of continued melancholic agitation. She is most demonstrative in her conduct and obtrusive in expressing her mental ailments. Her states of mental pain no longer well-up into explosive outburst—in impulses towards self-destruction, &c.; but, on the other hand, obtain continuous relief in motor agitation and querulous complainings. There is no pent-up energy here as in the case *H. T.*: it seeks and obtains relief in incessant garrulity.

There is here undoubtedly a frittering away of nerve-force from ill-conditioned cortical areas; but there is a vast distinction between such states and the genuine agony of mind apparent in acute melancholia. In the former, one is more struck by the continuous self-analysis, incessant introspection, and the fascination which the revelation of such states to others seems to possess for such patients: by the voracious appetite exhibited, and the maintenance of good bodily health, despite all this apparent distress. This accounts, in fact, for the chronicity of the case: for nearly four years this melancholic agitation has been maintained, and so far from exhaustion ensuing, the patient is well-nourished and robust. In fact, the painful mental state is far less real than one is inclined to imagine—the symptoms falsify the actual state: her utterances are but formulæ from frequent repetition.

We recall one patient, the subject of chronic mania, who would meet one every day with a lugubrious expression, and the remark:—"I'm going to be burnt to-day—I'm going to be burnt: they are building a huge fire in the park, and they are going to roast me on it." When questioned, she would enter into minute details of a horrible crime which she imagined she had committed, in which she had poisoned fifteen children with corrosive sublimate: would describe how she deceived the mothers: how she "watched the little brats partake of the poisoned meal, and wriggle about in their agony." Then she would grin maliciously or shake with laughter, ending with her accustomed formula—"I'm going to be burnt to-day."

Such cases impress us with the superficial nature of what often looks at first like profound mental pain; we must remember the peculiar cunning of the insane, who are always observant of the effect which they produce on the mind of the observer, their fondness for mimicry, extravagance, and distortion.

STATES OF MENTAL STUPOR.

Contents.—Stupor and Dementia—Etiology of Stuporose States—Stupor and Hypnotism—Stuporose Melancholia—Acute Primary Dementia.

By states of stupor we understand a suspension more or less complete of the emotional, intellectual, and volitional operations—a **suspension** in contra-distinction to an **abolition** of these faculties : the latter condition we denominate "dementia," a term which denotes the *absence* of certain mental faculties, through impairment or destruction of the mechanism whereby such operations are rendered possible. Whatever be the change whereby these faculties are suspended, whether as the result of pressure on the nerve-elements, the physical correlatives; or the result of the inhibitory effects of powerful sensorial stimuli; or temporary circulatory changes through vaso-motor influence; or exhaustive centric nervous discharges—the distinguishing feature is that of an arrest, transient or more enduring, of the intellectual operations, which may be **suddenly re-initiated** under an altered state of things.

As in cases of dementia, the abolition or impairment of these faculties has often notable accompaniments extending over a wide range of the cerebro-spinal operations, in impairment of sensation, blunting of the emotions, enfeeblement of volitional activities—so, in states of stupor, a similar impairment of the sensori-motor functions is apparent. These are the accompaniments, but the intrinsic nature of stupor depends upon the arrest of ideation and suspension of the intellectual operations.

The insane present us with every grade of these states of stupor, from cases of mild apathy, to depths of profound and persistent lethargy, in which the subject closely simulates the aspect of genuine dementia.

Mild forms of stupor often find their parallel in normal physiological life, as in the confusion of ideas which our waking moments are occasionally prone to exhibit. We knew a medical friend, accustomed to sleep heavily, thus partially awakened by the night-bell, receiving the message, dressing, and proceeding a considerable way upon his mission ere his destination and import of the visit were clear to his mind.

In these states, the perceptive faculties may correctly apprehend external things, but not their relationships to ourselves, and a state of transient stupor ensues. The state of post-epileptic stupor exhibited after a series of severe fits is interesting in this connection, as presenting a similar mental obnubilation to that seen in the cases of insanity to which we now refer. The vacant gaze, the dream-like look cast around, sufficiently indicate the torpor of the perceptive faculties; with the gradual re-instatement of the mental powers, semi-unconscious movements commence—fumbling of clothing, feeling or rubbing of the limbs, pulling about of furniture, incessant restlessness, and ill-regulated nervous discharges which usher in awakening consciousness. Such restless movements betoken the re-awakening mind, the re-energising of discharged centres, and find no parallel in states of stupor where the dormant intellect is shown by sluggish motorial reaction, fixation, and immobility.

We frequently observe, both in epilepsy and general paralysis, instances of suddenly assumed stupor, often of long continuance and not necessarily preceded by any obvious *motor* discharge or convulsion: undoubtedly, in these cases, there has been discharge of unstable grey matter from *sensorial* realms of the cortex.

Thus, a case of tabes dorsalis associated with mental symptoms has shown us this feature; whilst a similar case of insanity in a tabetic subject has, on the other hand, presented the post-convulsive stupor.

States of mental stupor may be variously induced thus—

(*a*) A mental shock, such as the sudden commotion caused by joyous or painful news in a high-strung sensitive subject, may have the effect of inducing such conditions of stupefaction: the acceptance of this fact is sufficiently attested to by such conventional phrases as "transfixed with horror;" "petrified by the scene;" "dumb with terror;" &c., &c.

(*b*) Nervous discharges from tracts of unstable cortex, as in epilepsy and general paralysis, lead to mental stupor through exhaustion of the centres so discharged: much here depends as to whether the centres so discharged have few or many sensorial or psychical correlatives.

(*c*) Other influences leading to exhaustive expenditure of force—exhaustive drains from the system—*e.g.*, phthisis—the vicious habit of masturbation.

(*d*) Acute forms of insanity are prone to be followed by stupor.

(*e*) Bromism, in like manner, whilst affecting the peripheral nerves, reducing the excito-motor functions of the spinal cord, and inducing torpor and sluggishness of secretions, leads to a loss of centric energy resulting in stuporose states: and other toxæmiæ have a like effect.

We must carefully distinguish these forms of stupor from the stupidity induced through obstruction of the nasal passages described as *aprosexia nasalis* by Gay; adenoid growths will here cause such obstruction to the lymph flow from the cranium as to induce much heaviness and stupidity, a vacant expression, a wandering gaze, a thick pronunciation, an open mouth from imperfect respiration, and dulness of hearing (*Victor Lange*).* The removal of such growths often leads to a rapid and marvellous change in the mental life.

Cases of stuporose insanity may be studied with advantage in connection with those exceedingly interesting conditions of hypnotism, which the researches of Mr. Braid, Dr. Carpenter, and more recently Heidenheim, Charcot, and Richer have revealed to us; and which now assume a more intelligible form as the phenomena become investigated by strict scientific methods. It appears that, in these artificially-induced states, every conceivable degree of suspension of the higher cerebral functions may be obtained, and the subject may pass from those light forms of induced reverie ("biological states"), through somnambulistic and cataleptic phases, into the more profound stage of mental lethargy.

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* *Centralblatt für Nervenheilkunde*, Marz, 1893.

The nerves of cutaneous sensibility, the nerves of special sense, the sympathetic centres in the medulla, may each be stimulated—the two former into a greatly exalted state of sensitiveness. The intellect may remain acute, but only on that train of thought for the time dominant: or the senses may, one or more, undergo notable blunting, and the reverie and state of **expectant attention** pass into dreaminess or profound sleep. Again, the muscular sense may be much exalted—automatism of an elaborate nature may prevail, reflex movements occur on suggestion, or tonic spasm ensue from excitation of muscles and tendons.*

Now, the states of mental stupor which prevail in the insane exhibit features which at times strongly suggest allied conditions of the cerebrum to those found in the "**biologised**" and "**hypnotic**" subject. In them, the suspension of the mental faculties and implication of sensation vary much in degree; may be *suddenly induced and as suddenly relieved;* in them, also, dominant ideas appear to prevail and cataleptiform states may be assumed, or still more profound torpor take the place of a half-dreamy state of consciousness. In them, also, the blunting of general and special sensation may be observed—analgesia, loss of taste, of appreciation of temperature, of sight, hearing, or of smell, noted in hypnotic subjects (*Landois*),† and in those conditions which pre-eminently favour what are known to be predisposing causes of hypnotic and trance-like states.

Mild stimulation of a special sensory tract, to the exclusion of others, notably of the *trifacial*, *optic*, and *acoustic*, as by the "passes" of the mesmerist, or the fixation of the eye on a bright spot above and near the eye to induce effort by convergence, or by soothing monotonous sounds—will, in many subjects, induce the hypnotised state. So, states of mental abstraction, where a monotonous impression or idea is the sole subject of thought, and where other impressions are voluntarily excluded, are conditions which pre-eminently favour states of induced hypnotism and stupor. It will be of interest to mention here certain prevailing features in the state of stupor and hypnotism, so as to indicate more clearly any physiological or psychological relationship existing amongst such groups of symptoms.

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* The earliest symptoms of hypnotism appear as the result of stimulation of the nuclei of the oculo-motor tract in the medulla: there is spasm of accommodation, restricted accommodative range, the *p.r.* approaches the *p.p.*: then stimulation of the sympathetic occurs with exophthalmos, widening of palpebral fissure, dilatation of pupil, quickened pulse and breathing.

† Landois and Stirling's *Physiology*, vol. ii., p. 269.

Mental stupor approximates more closely to the cataleptiform type of hypnotism, rather than to the truly cataleptic type. The subject is not, as in the latter state, accessible through the special or general senses, and suggestion through these channels fails to elicit responsive movements; but, on the other hand, it does appear that the mind is often the subject of dominant ideas imposed through external agency, and that the cataleptiform positions which the body and limbs may be made to assume, can be plausibly explained on the principle of suggestion through the *muscular sense.*

Yet, the patient in these states of stupor is not asleep, nor does massage or kneading resolve the rigid muscles which have assumed the cataleptic state. Herein, then, we see how the subject approximates to, and how far he differs from, the hypnotised individual. It must be added, that these clinical forms are by no means grouped together as suggestive of identical pathological states—the pathogenesis may be wholly distinct for each class.

The fixation of the limbs in artificially-imposed postures, would seem to indicate a dominant notion of the necessity for preserving such a posture, illustrating the obedience induced to external agencies whereby the will is subjugated—the subject's attention being reached principally through his muscular sense.

The greater depth of reduction in these states of stupor is attested by the fact, that suggestions by command fail to elicit such trains of ideas and resulting movements as the hypnotised will present. Closing the fists and advancing the arms of a hypnotised individual, and placing him in an attitude of defence, will often bring about fighting movements in reality (*Carpenter*); and muscular posturing will elicit the associated mental states of which it is normally the expression. This, of course, does not occur in cases of genuine stupor, or the more profound reductions of "acute dementia."

We see, again, simple forms of hypnotism in which the sole muscular anomaly consists in an inability to open the eyelids or the mouth: parallel states of mental stupor present themselves in which the same features prevail.

H. S. L., aged twenty-six, a married woman, with two children; the youngest, an infant, aged nine weeks, was weaned upon the outbreak of mental symptoms six weeks ago. No history of inherited insanity, neuroses, drink, or other vice. The labour had been natural in all particulars. On admission, she had a very vacuous expression: "stared round the room in a vacant manner: was wholly inattentive to what was said, and very rarely spoke. When questioned she usually remained silent, even though the query was repeated many times, and efforts were made to rouse her attention; or she repeated the concluding words of the question, or the words which she heard uttered by a neighbouring patient." She was emaciated and anæmic; her pupils widely dilated. The thoracic and abdominal viscera revealed no evidence of disease to physical exploration: but the bowels were torpid, and the tongue was foul and thickly coated.

After the operation of a saline aperient, patient was ordered a mixture containing 10 grs. of ammonio-citrate of iron, and 5 minims of liquor strychniæ in each dose (*bis die*).

The condition was one of painful stupor: the expression was melancholic and timorous, or one of complete stupor, in which she stood gazing vacantly into space: she was silent, but occasionally would give utterance to monosyllabic replies. Her habits were frequently negligent. She required feeding by hand, but was induced in this way to take abundantly. Her hands were cold and somewhat livid: all her movements were very sluggish.

About a week later, she became one day suddenly and violently excited—exclaimed aloud, "Cut my throat and let me die." Asked why she wished to die, she replied, "Because I am so shocked." Then she relapsed into her former abstracted, silent state, requiring continuous attention on account of her restlessness at night, her dirty habits, and her inattention to food.

A month after her admission her bodily condition had considerably improved: she slept better, but was still depressed and in a state of semi-stupor: the menstrual functions were in arrest.

Slow improvement took place in her bodily health, but amenorrhœa persisted for some six months, during which period the same treatment, alternated with iron and aloes, was maintained. She remained sluggish in her movements and somewhat depressed in spirits, but would freely converse about her state of health, and was eventually discharged as relieved to the care of her husband.

In such a case as the foregoing, we see the distinction between simple melancholic depression and the more *acute depression* often associated with stuporose states: whereas the gentle depression of the former induces apathy, disinclination for exertion, bodily or mental, and brooding silence, the latter may result in one of two conditions—either in the demonstrative expression of these painful states (*melancholia agitans*); or in a spell-bound stupor in which the organism seems, so to speak, petrified by its intensely painful mental state—the melancholy with stupor or the *stupidité* of French alienists.

Such patients are often completely dumb—their whole aspect that of intense stupidity; but, if you closely examine their features, you will observe evidence of painful emotion, or intense anxiety, of inexpressible grief, or perhaps a look of extreme bewilderment or concentrated astonishment.

Numerous patients, who have suffered from melancholy with stupor, have, upon recovery, recorded full details of their mental state: they are generally labouring under some frightful delusion, which utterly sways their consciousness and will: the outside-world may be a blank to them, and their whole mental life is subject to this all-absorbing delusion. Perhaps they imagine they have committed some terrible murderous deed, or that the end of all things is at hand: whatever it be, the attitude, facial expression, and demeanour indicate complete subjection to the engrossing delusion. This concentration of the mind upon one painful idea, which sways like an autocrat the whole organism, has been figuratively alluded to as a "crystallised delusion"—body and mind are crystallised around one morbid idea. Such patients often resist powerfully any attempts at feeding or other interference, and the refusal of food is sometimes most persistent.

Then come sudden, fitful gleams of mind at times; a rapid, hurried utterance, with as sudden a relapse into silence and self-absorption: or a sudden, mad attempt at self-destruction—an impulse, the direct result of the painful mental state. One should ever bear in mind this suicidal tendency in stuporose melancholia; it is a *constant danger* to be feared, and all the more since the apparent stupor is more that of *bodily activity*, and one is apt to forget that the mental state is often one of *intensely acute and painful strain*, most liable to explosive acts and impulses towards self-inflicted violence: all such suicidal attempts in this disease are frantic and determined in the extreme.

The following is an instance of permanent mental enfeeblement resulting from stuporose melancholia of long standing:—

H. T., short of stature, slight in build, and thin, was admitted at the age of twenty-six. She is a married woman, of steady, temperate, industrious habits, and was suffering from her first attack of maniacal excitement, the onset of which occurred a week ago. No predisposing or exciting cause could be ascertained for her attack.

Shortly after admission she became violently excited, and apparently in great terror and suspicion of all around. After a short remission of this excited stage, she became depressed, apathetic, and torpid in appearance. She would stand or sit in one position for hours, gazing vacantly before her—nor could she in any way be roused from this abstracted state. The catamenia have not appeared since her admission. Ten weeks after admission, her mental condition had so far improved as to permit of her attending Church service and entertainments; her bodily health also was considerably better. Shortly after this it is noted:—She is in a state of profound reverie—mental state apparently one of painful tension: fixed as though petrified to the seat or floor, her gaze is one indicative of intense self-abstraction and the prevalence of some delusional idea which dominates her whole life, and which now frequently issues, without any warning, in *sudden, impulsive, and most frantic attempts at self-destruction*, by throwing herself violently on the floor, or dashing her head against the wall. She would then lapse into a *cataleptic* state, in which her limbs might be made to assume any position for a lengthened period—her aspect trance-like, her expression indicative of intense and painful mental concentration: no vacuity, nor any appearance suggestive of dementia. Five months after admission, the painful mental state had subsided, but there was much stupor, with, however, occasional gleams of intelligence—transient recognition of her surroundings. She could not be induced to employ herself; was found one morning in a fixed attitude in the centre of the laundry wash-house, and on being questioned, said she was "at the Midland Station awaiting the down train." She was very pale and anæmic—there was amenorrhœa. She now takes iron and arsenic in mixture: subsequently altered to iron and aloes.

On June 6, 1883 —She cannot be persuaded to employ herself: still at times assumes fantastic, rigid, cataleptic postures, but is not now violent to herself—spasmodic outbursts of temper occur, when she is aggressive and destructive. Her expression now indicates a full appreciation of all that occurs around her, and judging from it, an absence of mental pain. Iron with cantharides and guaiacum ordered to-day.

June 11.—The catamenia have now appeared.

July 23.—A Faradaic current applied to the head daily for five to eight minutes.

July 31.—No benefit has resulted from Faradaism: to-day a constant current, from six to eight (Gaiffe's) cells, was substituted, one electrode to forehead, the other to occiput, the direction of the current changed by commutator several times, during five or eight minutes daily.

August 4.—Somewhat brighter in mind, cleaner in her habits, still idle: shows signs of indecency—exposing herself, and on being reproved threatens to slap the officer in the face. There is wide dilatation of the pupils; cataleptiform positions are still assumed, and long retained.

August 9.—Continued improvement: catamenia have occurred naturally again; has commenced employing herself at needlework: galvanism still continued.

October 11.—Constant current discontinued to-day: her attacks of impulsive violence and excitement, as also her cataleptic states, occur frequently. No improvement occurred subsequent to that above noted, and in June, 1884, it is recorded that she still has a fixed ecstatic look, is undoubtedly deluded, sudden, and dangerous. The case then assumed the character of ordinary chronic mania.*

Acute Dementia.—We have stated above that the extremes of stuporose states represent such profound lethargy as closely to simulate the aspect of genuine dementia: in fact, stuporose melancholia passes, by almost imperceptible gradations, into unequivocal dementia, and in several instances, we observed the melancholic stupor which characterised the onset of an attack pass into typical dementia.

In other instances, however, the reductions are from the onset so profound that a *primary* dementia occurs quite suddenly, and this represents the condition usually known as typical acute dementia. Acute **primary dementia** has been by some confounded with the stuporose form of melancholia—*melancholie avec stupeur*: by others, with simple stupor, *i.e.*, *suspension* of the intellectual operations apart from melancholic states, and not one of actual *abolition* of function.

Undoubtedly, however, there is a genuine acute dementia, in which the patient recovers to a certain extent, but invariably exhibits much impairment of his mental faculties ever afterwards. It is, of course, not suggested that the profound torpor, amounting in these cases to the appearance of utter fatuity, represents the degree of actual dementia, *i.e.*, of *destruction of function*: much of this is truly due to simple, though profound stupor—we can only judge of the amount of actual destruction of function upon the patient's so-called recovery. We find that this statement applies to all forms of dementia: all alike are liable to a certain admixture of stuporose states, which appear to emphasise the degree of abolition of mind—but which, dissipated by rousing the patient, indicate to us no such profoundly inactive condition as we should at first sight be inclined to predicate. In severe cases of acute dementia no such rousing can be induced, and upon restoration to more active function, the mind of the subject still fails to recall much (if anything at all) that has taken place throughout the attack.

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* In connection with stuporose states, Dr. Whitwell has communicated certain observations which appear to have an important bearing upon the pathology of some, at least, of these instances of stupor. Careful measurement of the blood-vessels at the base, by the graduated cone, leads him to infer the presence of a congenital narrowing of their lumen, associated also with a universal cardio-vascular enfeeblement. Dr. Whitwell's observations appeared *in extenso* in the *Journal of Mental Science*.

The following is an interesting case of this primary dementia, occurring in a young girl at the West Riding Asylum:—

A. J., who is now twenty-two years of age, was admitted seven years ago in a state of partial stupor. She had been regarded as imbecile from birth. Had never been able to read or write: although troublesome, she had not proved vicious, dangerous to others or herself, or of destructive habits. Had not suffered from epilepsy. Her family history was free from insanity, apoplexy, epilepsy, and phthisis. Patient had never injured her head. Quite recently it was recorded that she had become silent, gloomy, stupid, standing about staring vacantly before her; her habits were degraded—required compulsory feeding. At times it was recorded that she appeared in terror, and behaved as though she saw and heard imaginary objects about her. Upon admission there was much stupor: she remained perfectly mute to all questions, and her conduct did not lead one to believe that she appreciated either spoken or pantomimic language: a slight inarticulate cry alone escaped her. Facial conformation of low type—expression heavy, torpid. She showed a slight cataleptic fixity of the body and limbs at times: the limbs were cold and bluish. She was quite helpless, of dirty habits, and had to be clothed and fed.

The stupor rapidly passed off, and she was found to be a lively, good-humoured girl, very childish and imbecile, however, but able to speak in broken utterances. Became active, industrious, and cleanly: she was somewhat boisterous and excitable in behaviour at times.

In August, 1882, it is noted:—She has become heavy, apathetic, apparently demented; is negligent in her habits, never speaks, and requires all her bodily wants to be ministered to by others; she slavers at the mouth, extremities are cold and livid; catamenia have not occurred for six weeks. Is led about readily and exercised.

On the 11th of September she suddenly spoke to some patients, calling them by name, and remarked upon the beauty of some flowers near her. She then relapsed into her former heavy, drowsy state, and so remained until the 2nd of December, when she recovered her speech and mental powers as suddenly as they were annulled, and immediately began working in the wards. She was, however, flighty and excitable, and on the 9th of this month it is noted that she "is extremely excited and destructive, giving much trouble—is mischievous, disorderly, and violent"—for which attack she was treated by $\frac{1}{4}$ grain doses of hyoscyamin, and also by bromide with Indian hemp.

During the year 1883, she had repeated attacks of stupor which were characterised by their sudden onset and rapid relief; but, in all cases alike, a stage of maniacal excitement followed upon the stupor. In these attacks of stupor she stands about in a stooping attitude—motionless unless led; does not resist; head droops, the face is expressionless, and saliva runs from the partially open mouth; the pupils are widely dilated, the arms hang helplessly, the hands and feet are cold and livid. She has the aspect of one whose mental faculties are in complete abeyance. She remains where she is placed, and no voluntary movement is initiated—moves a step or two when pushed, and there remains motionless until again moved by others. She never attempts to feed herself, nor does she resist the efforts made to feed her, but swallows the bolus when introduced into her mouth: she is wholly inattentive to the state of bowels and bladder. The subsequent attack of excitement on return of partial consciousness was invariably of the same character—loud, hilarious, boisterous merriment, mischievous propensities, and occasional vicious conduct: she was also destructive of clothing, and wilfully destroyed glass and ornaments. Upon the reinstatement of her former mental health, she never could recall any experiences of her stage of stupor, although frequently questioned upon this point. The last attack of the kind occurred on the 15th of May, 1885, and her mental faculties did not clear up until the 20th of November, a period of six months.

In such cases, we do not find the subject prone to sudden, wild outbursts of maniacal or melancholic frenzy, such as we found prevailed in stuporose melancholia—to active and desperate attempts at self-destruction, *followed by as sudden a lapse into the stuporose state.* The condition of mind is distinct in the two affections: in the one (the melancholic) it is strained and ever prone to explosive outbursts, in the other (the demented) *too feeble to initiate any such attempt.*

The maniacal outbursts characterising the last case detailed, were really upon the road towards a restitution of the normal state of mental health: as the stupor lifts, so the maniacal reductions come to the front, ere the subject is restored to her former self. And yet, *not to her former self*, for a passage of her life, as we see, is completely obliterated, and the mind is a blank to the events of each attack.

The **apathetic passivity** of such cases also contrasts strongly with the resistance, and often violent struggling, offered by the subject of melancholia cum stupore. The blank, stupid, idiotic stare, and the utterly demented expression, are likewise very different from the aspect presented by the latter affection, where mental tension is very evident in the pained look, which sometimes is varied by gleams of transient ecstasy. **Cataleptic fixation** of the limbs is a frequent, but by no means invariable, accompaniment of acute dementia.

Some subjects of this disease are, according to Dr. Blandford, in incessant movement: "One girl used to snap her jaws together for days at a time, and then changed to wagging her head from side to side." These are the less profound instances of reduction.

The heart is feeble, and there is great torpor of circulation—the hands and feet being cold and livid to an intense degree. Such

STATES OF EXALTATION.

Contents. —Maniacal Reductions—Failure of Attention—Enfeebled Synthesis—Transient Delusive States—Exalted Sense of Freedom—Impulsive Conduct—Nocturnal Crises—Seclusion Fosters Hallucination—Sexual Illusions—Stadium Melancholicum—Enfeebled Imagination—Bodily Symptoms—Periodicity of Maniacal Phenomena—Acute Delirious Mania.

Viewed from the clinical aspect, cases of mental depression chiefly impress us with the prevailing feature of **mental pain**; although, as we already have seen, mental pain is by no means an *essential* element in states of **mental depression.** The latter term to us connotes far more than simple melancholic pain, since we regard all cases of simple intellectual torpor, morbid apathy, and states of simple stupor (all of which may be devoid of painful emotional states) as comprised under the category of states of mental depression. In like manner, although we may take the prevailing emotional tone, the exuberant flow of thought, and the general objective indications of maniacal excitement respectively, as characterising states of mental exaltation, we must remember that any one of these indications may be variably pronounced, or even suppressed. We should, therefore, carefully define to our minds the connotations of these respective terms—**exaltation** and **depression**; and we shall then learn that the more arbitrary, narrow, and exclusive our definition, the less readily do we perceive that the one is the converse of the other; and that the freer our definition becomes, so as to embrace all mental operations within its limits, the more readily do we find in the infinite varieties of both classes, states in the one which are the exact antitheses of states in the other.

It is, however, of far greater interest to recognise, in the morbid process which underlies these states of mental exaltation, that the process of reduction is usually more sudden in its onset, more rapid in its course, more intense, and the level reached always lower than that of simple mental depression.

Mania is far more prevalent than other forms of insanity amongst the less civilised races of mankind; thus the percentage of mania amongst the Kaffir race is given at 67, and melancholia is distinctly rare, and not acute (*Greenlees*). In reference to this interesting fact Dr. Greenlees writes:—"If we consider the theories of those who maintain that while mania represents a loss of the lower developed strata of the mental organism, melancholia indicates an absence of the higher and latest developed strata, then this prevalence of mania amongst natives of low developed brain-functions goes far to prove this theory."*

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* "Insanity amongst the Natives of South Africa," by T. Duncan Greenlees, *Journ. of Mental Science*, vol. xli., p. 72.

In states of mental exaltation, we also trace the same failure in object consciousness, with the corresponding rise in subject consciousness, which states of mental depression present, but how different are the features of the two viewed in contrast.

Here we have in the welling-up of feeling, pleasurable emotions in place of painful states; a general sense of well-being, exuberant joy, excessive hilarity, an overflowing of the spirits in generous impulses, an egoistic self-confidence—all strongly contrasting with the grim foreboding of coming evil, the gloomy aspect of the present, the sorrows of the past, the sense of the subject's helplessness before an encroaching and malign environment. So also in the rapid flow of thought—disconnected, incoherent as it is—expressed in rapid utterance, associated with restless movement, energetic pantomime, and a sense of utter lawlessness (often issuing in reckless conduct), we have a striking contrast to the sluggish ideation, enfeebled imagination, apathy, paralysed energy, and restricted movement of the melancholic.

Yet, fundamentally distinct as these mental states would appear to be, we have little doubt that the process of reduction is the same for both; but in maniacal states the dissolution is to a **greater depth**—the difference is one of **degree.**

All maniacal conditions are pre-eminently distinguished by a failure of attention or of the capacity for **serial thought,** and a rise of the purely sensuous in place of the intellectual operations—in fact, the latter are enfeebled, and the emotional elements are aroused; and, as before indicated in cases of *depression*, the intellectual sphere presents the negative, and the sensuous the positive results of the reduction.*

Since seriality of thought requires high nervous pressure—a high-tide of the nervous wave to force the ultimate ramifications of the cerebral cortex—so here in mania we must recognise an ebb of this cerebral tide, corresponding to the lowered plane of psychical activity; for the activity which we recognise in the excitement of lower levels is one of disorderly ungoverned license, indicative of the removal of the influence of higher controlling planes.

Of the three laws of association of ideas which regulate our normal mental operations, the law of association by similarity embraces operations of a far more abstract nature than is the case with that of association by contiguity, and the same may be said of the latter as contrasted with the process of association by accident or incongruity. It is the more abstract representative processes of **association by similarity** which are first involved in maniacal reductions—the less abstract presentative-representative processes not being so far involved. This fact explains much of what we observe in the maniac's conduct; his perceptions are crude, and his notion of the essential utility of objects around him is frequently at fault—the result is often comical, but at other times it is disastrous to himself or others. Thus we may see such a subject trying to put his coat on by thrusting his legs into the sleeves, mistaking the garb for a pair of trousers; here we observe that the association by similarity suggests to his mind only imperfectly the utility of the garment. In fantastic attempts at decoration, in the wanton destruction of objects around, in the tearing-up of clothing and bedding, in the swallowing of garbage, and in some of the most repulsive tendencies of the maniacal, we must recognise the failure of consciousness implied in the imperfect operation of association by similarity, and not refer such acts, as is so often done, to sheer wilful mischief. The imperfect operation of the same law is seen also in the remarkable rhyming tendency presented by some, viz., that of stringing together, in verse, numerous lines utterly devoid of sense, but in which a well-regulated rhythm pervades the whole, and each line accurately rhymes with its fellow. Numerous instances of the restricted operation of this law will occur to any one familiar with the vagaries of the insane.

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* Just as the sensitiveness of the retinal field declines progressively from one acutely sensitive spot outwards, until impressions received upon the outermost peripheral areas are more and more dimly perceived and eventually fade, and just as we tend by concentrating our attentive gaze on a fixed point to limit the field of conscious perception and to *press out of consciousness* those obscure faint excitations of the outer field of vision: so with the mental field, as the serial line of thought becomes restricted, as object-consciousness fails in vigour, so there crowd into our conscious life those obscure and multitudinous impressions which are always present, but are, for the most part, excluded from the immediate gaze of the mind's eye.

It is in this failure of the highly representative processes that we must also learn to trace the early origin of those deluded states to which mania and melancholia tend; for we may well see how by their failure sense-presentations are not so likely to have their falsifications corrected—an appeal to a higher tribunal is not permitted. **Transient delusions** thus characterise the maniacal turmoil throughout its history; they are often but indications of a **want of balance** established between contiguous groups by discharges from higher controlling realms, leaving, so to speak, certain islets "flooded;" such perversions are of a very **recoverable** nature. As contrasted with the fixed perversions of monomania, Griesinger thus alludes to them:—

> "But if these two forms in their extreme degree are so utterly distinct as mutually to exclude the possibility of mistaking the one for the other, still observation shows that in mania itself such delirious ideas of self-importance are by no means unfrequent, which certainly ought not to be regarded in an ontological sense as 'fragments of monomania,' as phenomena of quite another affection which in this case present themselves in a very simple form, but as the result of the primary mental condition. . . . They (*i.e.*, the delirious ideas) share in the tumult and precipitancy which agitate the motory sphere of the soul-life;

they become so confused in their hurry, and pursue each other so rapidly, that they have no time to become fixed or to dwell in the mind." *

An "over-action on lower planes," as Dr. Hughlings-Jackson would term the state to which we allude, characterises these maniacal states in the intellectual sphere, revealing a profound failure in object-consciousness. Ideas arise in extremely rapid sequence, often as the mere result of casual or accidental association, the subject being swayed by every passing incident. Every degree of incoherence may present itself according to the rapidity of the cerebral processes at fault; yet, in all the simpler forms of mania, a temporary command of the attention can be obtained by an authoritative gesture, command, or other artifice, when a perfectly coherent statement may be elicited, the memory exhibit normal vigour, and reason momentarily assume her sway; then they pass back again into the wild turmoil of disconnected ideas and strange mental combinations. It is in the changeable, fleeting nature of the mental images that we hope for the patient's complete restoration to mental health.

It must not be imagined that *all* states of mental exaltation imply the tumultuous career of ideas above described. Ideation is always quickened by unnatural vigour—the images become more vivid; but a superficial coherence may be observed which enables the patient to hold rational converse, to employ cogent argument, raillery, sarcasm, or wit. In the simplest types of mania, slight garrulity, a restless movement, and rapidly-varying mood may alone betray the mental disturbance.

If we turn our attention to the sphere of subject-consciousness, we find here in the majority of cases a rise of the pleasurable emotions and an unwonted sense of freedom, undoubtedly engendered by the coincident, unrestrained activity dominant in the intellectual sphere. The high-tide of the emotional wave reacts on the intellectual sphere—the exalted self-feeling issues readily in action, or begets with equal ease notions of power and self-importance—*transient* delusive concepts, which rise as new creations, answering to the prevailing mood. The exuberance of feeling usually expresses itself in goodwill to all mankind—in a universal optimism, which often issues in schemes of philanthropy as impracticable in their nature as they are transient in their duration—in extravagant and ludicrous proffers of patronage to science and arts; or the mood may vary from this to one of supreme arrogance, in like manner conjured up by the exaggerated self-feeling; and the subject may announce himself to be some mighty personage, and assume a defiant, threatening, or savage aspect. We observe that these reductions in maniacal states bring the subject to a more **automatic or instinctive level;** impressions received from without are liable to issue in immediate action—mature deliberation no longer characterises the mental operations, but a state of exaggerated mental reflex; in like manner, the animal passions and instinctive desires, *uncovered, as it were,* spring into life and show an unregulated activity—**impulsive conduct,** therefore, is especially prevalent in states of mental exaltation, and the maniac may be destructive, violent, blindly impetuous, or dangerously homicidal, or react to any of the grosser animal passions and instinctive desires by which he is swayed. It is this **instinctive** level to which maniacal reductions attain that characterises the features of mania as contrasted with melancholia; and, as will be stated later, the prevalent psychoses of earliest childhood (prior to the evolution of higher grades of mental activity) are peculiarly **instinctive** forms of derangement—mania, not melancholia, and wholly devoid of delusional accompaniments.

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* *Op. cit.*, p. 274; see also Theodore Kolle on "Variability in Delusions"—*Allgemeine Zeitschrift für Psychiatrie*, Bd. xlix., Heft 1 und 2.

Nocturnal Crises.—The insane are peculiarly liable to be affected by those cyclical conditions which are recognised in the healthy individual, and thus the periods of waste and repair, embraced by the day's labour and the night's rest, are shown in their case also. The phenomena of nocturnal crises, and the periodic character of their excitement, are very notable and well-recognised facts. The daily routine of work may be passed through in a quiet, orderly manner; obedient to the "law of the room" and the injunctions of the nurse, tractable and reasonable in conduct, the insane may exhibit no outward indication of mental anomaly until more closely examined; yet, invariably, as night approaches, they pass the hours in loud, boisterous excitement, shouting, singing, incessantly chattering, replying to imagined voices, restlessly wandering about, or beating the doors or shutters of their room. During the day, the association with their fellow-patients, application to their various duties, and the general discipline of their immediate surroundings may have just sufficed to engender that control over their conduct to which there is now no incentive. The seclusion and quiet of their rooms, the release from all imposed reserve, permit of that wane of object-consciousness which is invariably followed by a rise of subject-consciousness. This nocturnal crisis must be regarded as the outcome of those rhythmic changes, which, in a normal state, should issue in *sleep.*

Such reductions, however, are but partial, spasmodic, and limited to psychical processes only—whereas in sleep the whole excito-motor apparatus is more or less deeply involved. In sleep, object-consciousness quickly, even *suddenly,* succumbs; subject-consciousness goes *more slowly,* and the more profound depths are not usually reached for *an hour,* or even longer; the reflex excitability of all the nervous centres (spinal also) is reduced—the organic functions are lessened. In these cases of nocturnal excitement, however, the effect of this periodic lapse of consciousness is to call up more turmoil at lower levels; all those subjective states arising from epi- and ento-peripheral stimuli, or centrally initiated, become the subject-matter of the mental view; all those disconnected and simultaneously originating ideas which crowd the mind, and which, in healthy waking states, are reduced to serial, orderly thought, now run riot—and beyond this, hallucinations of the special senses prevail.

Seclusion tends to foster **hallucination**; this fact, so well recognised amongst the criminal community in prison life, is especially true as regards the insane. It is to the existence of hallucinations that we must largely attribute the insomnia and noisy outbursts; and it is an undoubted fact that many such cases, judiciously selected, are benefited by removal to an associated dormitory, and thus nights previously passed in noisy excitement become intervals of repose and quiet.

Such hallucinations, often peculiarly vivid, fascinate the mental vision, and according to their nature call forth corresponding results—the patient may be passionate, wild, threatening and defiant, abject with terror, shouting in alarm for succour, joyous, exultant, or in boisterous merriment; every phase of emotional life may present itself as the hallucinations vary, and he enacts his little drama alone.

In general paralysis these nocturnal orgies are frequent—noisy restlessness, with or without hallucination, accompanying the later stages of most cases.

Hallucinations are not infrequently referred to one side only; but, more frequently bilateral, they may alternate from side to side (*Toulouse*); or, being quasi-bilateral, may vary in their character, assuming thus, in auditory hallucinations, a hostile, threatening, abusive tone on one side—a consolatory, cheering tone on the opposite side (*Magnan*). These antagonistic hallucinations are of extreme interest. In 39 cases of unilateral hallucinations observed by Toulouse,* hearing was involved in 26, sight in 7, sight and hearing in 4, sight and touch in 1, and in one other case touch was also affected. Of the 26 unilateral hallucinations of hearing 23 were strictly unilateral, 1 case alternated from side to side, and in 2 others the bilateral antagonistic features alluded to above were evident. It is a well-known fact that unilateral hallucinations of hearing may appear in focal hemispheric lesions. Thus, in an epileptic subject with right brachiofacial paralysis, aphasia and lateral homonymous hemiopia, and, on another occasion, associated with loss of sensibility, there was the accompaniment of auditory hallucinations on the *right* side (*Pick*).† Unilateral hallucinations of hearing and, vaguely, also of sight occurred in a case of alcoholism, where Jacksonian epilepsy (with other symptoms indicating focal lesion of the right hemisphere) existed—the auditory hallucinations being restricted to the *left* side.*

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* *Gaz. d. Hôp.*, May and June, 1892.

† *Neurologisches Centralblatt*, No. 11, 1892.

In many, the hallucinatory phenomena are *recognised* by the patient as having no *real* objective origin, and yet they will be fostered by the fascination which they entail; especially is this the case with **sexual illusions,** which are frequent in the female sex, and which, it is probable, are very largely entertained and fostered. Cases occur where the nocturnal reductions having been recovered from, the patient is filled with remorse or shame, or accuses certain individuals of criminal conduct, accompanied by threats of retribution and violence; each night the phenomena recur, attended by erotic excitement, and each returning morning they form the incentive to indignant protest or violent conduct.

To many again, the reductions entailing rambling, disconnected thought and garrulity, are in themselves a source of pleasure—easily controlled when the patient is brought into association.

The exhaustion which often follows such nights of excitement and, possibly, the accumulation of decomposition and waste products in the blood, induce in many prolonged sleep throughout the day.

Mania.—The incubative period of mania is but a record, in most cases, of a very gradual declension in mental vigour, not perhaps at all apparent to the friends, but sufficiently evident to the subject himself. Intellectual operations become more laborious than usual, thought is sluggish and tends to wander (attention being fugacious); strange and unusual lapses of memory occur—the patient is "absent" and forgetful. All mental operations are not only difficult and tedious, but are followed by weariness and ennui, and a gloom overspreads the mind, for which there is often no obvious cause. At night the subject is restless, obtains but little sleep, and awakes unrefreshed, with gloomy forebodings, and a disinclination for all forms of exertion; in fact, a frittering away of nervous energy has brought him into the first stage of his malady—that of simple melancholic depression, to which all the foregoing remarks we have made with respect to simple melancholia apply. This, the first stage of his reductions, is the **stadium melancholicum** which precedes all forms of mental disease. It is not implied by this that the subject *necessarily* exhibits such a stage in all cases—although, undoubtedly, many cases cited of sudden onset of excitement without previous warning, are instances of a defective observation on the part of others. The **absolutely sudden onset** *of maniacal symptoms* does occur at times, as in instances of **epileptic insanity** from sudden and severe discharges: nor is there any *prima facie* reason for supposing that such serious and sudden reductions should not occasionally be induced. We must, however, regard the melancholic stadium as the usual feature, and the sudden onset of mania as quite exceptional. This premonitory stage is of most variable duration, ranging from days to weeks, or from weeks to months; it may pass off under favourable circumstances, and again recur as former conditions of life are resumed.

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* Toulouse, *loc. cit.*

Then suddenly, and often quite unexpectedly, comes a marked transformation, signalising the *maniacal* reduction. The gloom and despondency appear to be lifted off—reticence and brooding are replaced by sociability and vivacity: a strange light gleams in the eye; an animated expression replaces the pained and stolid aspect; the moods are mobile, and an exalted, pleasurable self-feeling pervades the subject. His thoughts, no longer under painful restriction, flow in unlicensed freedom and in unwonted rapidity, reproducing the symptoms of early alcoholic intoxication. The patient is garrulous—obtrusively so: talks about his own affairs; is confidential and communicative to utter strangers; is egoistic, makes profuse offers to befriend all around him; is energetic in his movements, incessantly restless, and rapid in his utterances. If we test his individual faculties, we may find his memory fairly intact, or partially obscured, upon events occurring during his maniacal attack; his attention is commanded with more or less difficulty, according to the intensity of the excitement (depth and extent of reduction), but in all simple forms of mania it is readily brought under control; yet only to lapse the next moment before the tumultuous flow of incoherent thought. If we leave him to his own devices, and listen to his rambling speech, we discover that fragmentary condition of language which attests the want of coherence of ideas—a weakening of that synthetic process which renders intelligent and rational thought possible; a cohesion of ideas still is apparent, but it is that of the *trivial* associations chiefly, and suggestive movements, utterances, or other impressions presented casually by those near him, will often blend promiscuously with the subject-matter of his thoughts, in the most grotesque and unregulated manner; and, as we have before explained, the *seriality* of thought becomes impossible. Every degree of incoherence may thus present itself, from the mildest occasional rambling, to utter incoherence, where speech is quite unintelligible, as in the deepest reductions of typho-mania or acute delirious-mania. The patient is reduced to a more automatic level; his actions are more instinctive than volitional, just as his ideation is more reflex in its arousal and expression. A slave to every passing impression, to every casual thought, to every emotional incitant, his conduct is wholly unpredicable, subject to no rule or means of calculation. The maniacal subject is not imaginative, in the proper sense of the term; at times we find what savours of imaginative vigour, but all such gleams are superficial, transient, and accidental; the strangest combinations of ideas must necessarily prevail at times, and produce this apparent imaginative turn—and the unexpected scintillations of wit which characterise certain maniacal subjects. Like the child, his imagination is feeble and inchoate, and, like the child, too, his flimsy fancy wanders aimlessly, and replaces the truly synthetic, creative operations of the imaginative faculty. Judgment may be perverted upon certain points, but is just as frequently unaffected; in fact, the mental faculties exhibit only such derangement as would occur from the excessive activity of the maniacal process—a transient confusion or partial suspension due to the rapid flow of ideas. By this we do not mean that the subject of mania does not suffer from delusions. Delusions are a constant feature in maniacal excitement; but they are extremely transient, rapidly varying in their nature, and changing with the ever-changing mood; their superficiality declares itself in their continuous displacement by fresh delusive ideas, for they do not remain permanent, as in the false conceptions of so-called monomania. Their origin can be attributed only to the confusion and tumult of ideas occurring with the emotional background of exaggerated well-being and unnatural egoism; in fact, on the prevailing tone of the moment seems to depend the character of the false belief entertained. The following case illustrates what we have just said:—

G. R. L., aged twenty-one, single, by occupation a dyer. A young man of moderate height, muscular, pale, and anæmic, with an icteric tinge of skin: a very retreating forehead. Last year he had suffered for some five months from a similar attack to his present seizure. An uncle of patient's was depressed, but no further clue to heredity was obtainable. He had suffered from convulsions during dentition, but his health had proved satisfactory up to his first attack of mania. He had been somewhat intemperate in his habits. He was in a state of continuous maniacal excitement, shouting aloud, singing, laughing, and gesticulating frantically. At first he did not sleep well, and was noisy through the night; chloral was given with good results. Through the day he was boisterous and unruly, rambling incoherently, and destroying his clothing. He raps the walls with his knuckles—calls out in imperative tones to imaginary individuals with whom he holds converse; but calms down on being spoken to authoritatively, and condescends to give certain information respecting himself in a grandiose style and a pompous voiçe. In almost the same breath, he declares himself to be the "Prince of Wales—the Prince of Peace—Lord of lords and King of kings; his mother is the Duchess of Kent." He is fully aware that he is in the West Riding Asylum, and gives correctly the date of the month and other particulars. He assumes fantastic attitudes, which symbolise his prevailing feelings for the time; struts about in pompous style, throws himself into an attitude of wrapt attention; or with lowering brow and clenched teeth apes an aspect of rage and defiance; then, as suddenly, with a lordly wave of the hand and gracious smile, he addresses those around him by dignified titles, the very next instant to lapse into the clown and turn summersaults about his room. At one moment he announces himself as General Gordon—at another he is Sir Garnet Wolseley, and by tone and gesture assumes a military bearing. These rapidly varying delusional states, the one supplanting the other, all indicate the egoistic sentiments of the mind, the overflow of animal spirits, the superabundance of energy finding free and ready expression in incessant movement, pantomime, and speech. From the very outset, his habits were negligent and degraded, and he was early found to be addicted to masturbation; his gestures, also, and expressions often indicated a sexual excitement. When referring to insanity at the adolescent period, we shall find this arrogant and egoistic state of mind to be often associated with habits of masturbation.

In the course of six months, this patient's excitement entirely disappeared, and he was able to give a fair account of his feelings, affirming that he believed himself, throughout the attack, to be some great personage with military functions.

The exuberant swell of feeling, and the torrent of disconnected ideas, may express themselves in continuous garrulity, in noisy chattering, in threatening and abusive tones, in laughter, singing, or loud shouting, with corresponding pantomime and almost ceaseless activity; or the feeling of unusual freedom and energy may find relief in destructive tendencies—smashing of glass, breaking of furniture, tearing of clothing, or, perhaps, in violent aggressiveness.

The rapid alternations of disposition are peculiarly striking; the surging of the emotional wave is followed by an ebb, only to reappear in other forms, so that intervals of calm may find the patient even reticent, despondent, or abjectly miserable, until some trifling cause lights up the flame afresh. These intervals of depression are in nowise different from the melancholic states; in fact, *it is but a step* from the maniacal to the melancholic stage. At this instinctive level, the patient instantly reacts to the most trivial excitant, with utter disregard to decency; peripheral irritation may thus induce open and shameless masturbation, or nymphomaniacal states may render the subject of either sex repulsive in the extreme. In like manner—dirty, degraded, negligent habits arise, and depraved appetites spring into life. Sensorial disturbances, in the form of illusions and hallucinations, are of very frequent occurrence in mania; and, at times, it becomes difficult to engage such patients in conversation, so intent are they listening to these phantom voices, or busy shouting aloud their replies; or, whilst talking to us, the rapid turn of the head, the hurried gesture, the interposed exclamation, or irrelevant remark, indicate these sensorial phenomena.

Bodily Symptoms.—Although, in a certain proportion of cases, especially in alcoholic and senile subjects, and in the maniacal excitement of general paralysis, we note considerable injection of the vessels of the head and neck with a suffused aspect of the face—the great majority of maniacal subjects undoubtedly exhibit *marked pallor of the face*—the skin generally being also of yellowish tinge, unctuous feel, and foul odour. The pulse is small, somewhat frequent, and the heart's sounds are often muffled. Griesinger[*] speaks of the heart's sounds as being indistinct during the paroxysm of excitement, and becoming clear during moments of calm; during the maniacal paroxysm also we learn from Dr. Clifford Allbutt[†] that the **optic disc is anæmic,** becoming, in a few days subsequent to the attack, suffused and obscure. The spasm of the retinal vessels, presumably present in these cases, appears to us of great importance in revealing the true nature of the maniacal process as distinguished from states of mental depression.

The tongue is often coated and foul; the bowels are torpid; whilst the appetite, sometimes indifferent, is more frequently exalted and often insatiable. It matters not how well the patient takes his food—incessant activity and continued insomnia are sure to result in loss of body-weight; in most instances great emaciation prevails—the face assuming a pinched appearance, the features are sharpened, a dark areola surrounds the eyes, the eyeballs are sunken, yet restlessly active and mobile. The re-instatement of a well-nourished frame is a rapid process *cet. par.* upon cessation of the maniacal symptoms. There is frequent interference with the menstrual function, during the course of mania. Despite the incessant motor agitation and excitement, the **body temperature is apyrexial** and normal.

Periodicity.—During the course of maniacal excitement, a remarkable periodicity is often noted in the exacerbations and remissions which occur. We have already alluded to the nocturnal crises which are very prevalent, and we may now draw attention to diurnal variations of excitement and calm occurring upon alternate days, and to which the attention of the nurse is often attracted—an observant nurse will often speak of such a patient's "*quiet day*" or his "*bad day,*" referring to this strange alternation. We quite as frequently find the subject alternate between mental exaltation and depression from day to day, and this ready transition from one form to the other appears to us of the highest importance for a proper comprehension of the pathogenesis of these mental states.

The menstrual molimen is especially prone to arouse in these subjects an exacerbation of excitement, so that a monthly periodicity in these maniacal outbursts (with more or less complete remissions intervening) is by no means infrequent. Frequent relapses occur in certain subjects, apparently attributable to the vicious habit of masturbation—maniacal reductions and stuporose states being often readily incurred.

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[*] *Op. cit.*, p. 288.

[†] *The Ophthalmoscope in Diseases of the Nervous System.* Dr. Allbutt's observations are based upon the examination of as many as fifty-one cases of mania at the West Riding Asylum.

Prognosis.—Acute mania occasionally proves fatal; the explanation of such an untoward event is the presence of some intercurrent affection such as pneumonia, the exhaustion of phthisis, or the association with septicæmia, as in certain forms of puerperal mania. In fact, puerperal mania occurring as the sequel to exhausting hæmorrhages—*ante or post partum*—is a very grave affection and not unlikely to issue in a fatal termination. If we exclude the mania symptomatic of epilepsy, general paralysis, and alcoholic insanity, we may state that simple uncomplicated acute mania is a remarkably *recoverable* affection.* Exceptions, however, occur even in uncomplicated mania, where, from the neglect of early and judicious treatment in greatly debilitated subjects, death ensues.

Far more frequent than a fatal termination is the establishment of chronic mania, in which a permanent mental enfeeblement results, associated with recurrent attacks of excitement. Especially ominous of this termination is the return of physical health and vigour, the re-establishment of the nutritive functions, the tendency to *enbonpoint*, or actual obesity in some cases, with no corresponding improvement in the mental state, but persistence of the maniacal reductions, and a general mental enfeeblement. States of mental enfeeblement including chronic mania, consecutive dementia, and the monomaniacal perversions represent the several modes of unfavourable termination when recovery does not ensue. The mode of recovery varies much in individual cases; it may be abrupt—so abrupt that in the course of twenty-four hours the patient may emerge from a state of acute excitement into one of coherence and perfect calm, with, it may be, a slight confusion alone recognisable; such terminations are not desirable, and almost certainly issue in relapse which may recur over and over again ere permanent recovery is ensured.† The more reliable process is a steady return to former levels of mental life; a process of gradual evolution, broken, it may be, by slight reductions in which excitement temporarily increased invariably leads to subsequent stages of clearer consciousness—a process in itself both instructive and reassuring. Savage affirms that the more prolonged the initial melancholic stage, the longer is the second stage of excitement likely to be; the longer these two, the less hopeful the prognosis; and the greater the amount of excitement, the greater is the tendency for the patient to pass into a condition of depression afterwards.

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* Reference to the table of results on p. 221 will illustrate this fact, bearing in mind that the percentage of deaths in acute mania (13·4) includes uncomplicated as well as all complicated cases.

† Such abrupt terminations of maniacal excitement are apt to take place on the occurrence of some acute bodily affection—*e.g.*, pneumonia, erysipelas, the appearance of carbuncles or boils—by some regarded as suggestive for a derivative system of treatment.

Age is a factor of much importance as regards the question of recovery; the younger the subject, the more likely is he to recover. We do not even exclude the mania of adolescent insanity here—so far as it relates to the female sex; but, should the psychosis characteristic of adolescent insanity occur in the male, the prognosis is far more unfavourable.* So, likewise, for recurrent mania—each occurrence serves but to re-establish a still greater tendency to maniacal outbursts, the attacks becoming more frequent, the intervals shorter, and the enfeeblement of mind steadily progressing.†

Acute Delirious Mania.—This, the **délire aigu** of French writers, represents the most profound maniacal reductions which we meet with, just as simple mania connotes the symptoms of the milder reductions. The disease is often most sudden in its onset, and frequently appears to follow upon some moral cause—shock or fright. This, however, is attributable to the special predisposition of the subject, evidence of excessive instability being in most of these cases afforded by the history. It differs from ordinary acute mania in the **intensity** of the process, the extreme reductions in object-consciousness, the **absolute oblivion** in most cases to all around, and in the *rapid course* and *frequency of a fatal termination*. It is quite exceptional for a case of acute mania to prove fatal; in fact, unless the individual is much debilitated prior to the attack—suffering from some exhausting ailment, such as phthisis—or when it is the sequel of exhausting hæmorrhages, as after parturition, we augur well for our most wildly-excited patients. A case of uncomplicated acute mania usually means a certain and rapid recovery. Not so, however, in acute delirious mania; here the outlook from the first is most ominous, and the gravest prognosis must be given. The tongue is dry and brown; the lips and teeth become covered with sordes; food is often most peristently refused, and violent struggles made upon attempting artificial feeding. The patient is usually quite oblivious to our intentions, and obstinately resists all we do for him. He presents a pitiable spectacle, is unsteady on his feet, totters and sways from sheer muscular debility and exhaustion, and trembles in his limbs. His utterances are a broken strain of completely unintelligible jargon—the **incoherence being absolute;** the lips tremble, and speech becomes eventually a mere babble of inarticulate sounds, interspersed with sobbing respiration. Sleep is entirely abolished, muscular wasting rapidly proceeds, and in a few days he is so prostrate that he lies helplessly on his back, unable even to assume the sitting posture. He now represents the condition often described as **typho-mania.** The temperature is always raised more or less, sometimes to 102°. The urine may be scanty or suppressed; it may pass involuntarily, as do the stools. If intelligently-directed treatment be not early adopted, a rapidly fatal termination ensues; and even under the most favourable circumstances, the struggle to bring the patient safely through the storm is an anxious and prolonged one.

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* See under *Insanity of Puberty and Adolescence.*

† *Cf.* p. 250.

Cases of Delirious Mania.

A. H., a married woman, forty-seven years of age, was admitted after excitement of seven days' duration. She had suffered from mental derangement some four years ago, attributed by her friends to her son's running away from his home; was under treatment at an asylum. A week ago the same son again decamped from home, and the mother's distress culminated in the present seizure.

No history of insanity, neuroses, drink, or other vice in her ancestry. She was a very emaciated subject, of pallid, sallow, pasty complexion, with dilated malar venules; there was a strongly marked divergent strabismus. She was suffering from considerable bronchial catarrh; the pulse was 148, regular, but very small and feeble. There was extreme anæmia—the jaws were edentulous, the abdomen sunken, the left hypochondrium rather tender, no splenic enlargement. The genito-urinary system appeared normal.

She was restless, excited, trying incessantly to leave her bed, and talked continually—uttering ejaculations such as, "Oh! my God! what shall I do!" She was extremely prostrate, fainted on admission, and nourishment had to be forcibly administered by means of the stomach-tube.

She did not sleep the first night, and next morning was in a condition of acute delirious excitement, rolling her head about in bed, tossing her legs, fumbling with the bedclothes. All her utterances were irrational and completely incoherent. When asked why she came here, she remarked—"To drink! it makes great distinction in the sex of your business—Follow me—I have been in the feminine of giving drink—Oh! oh!—I am receiving gentlemen, not you—Remember the sex—The feminine discretion of the place of my lips." She refused food, "because it is so abominable, it is so obstinate to the effect of my heart." She was not violent, and her tone was elated, not depressed.

Essence of beef with milk, eggs, and port wine (6 ozs. daily) were ordered; 10 grains of the citrate of iron and quinine, *bis die.* Two days subsequent to admission it is noted—"Exceedingly prostrate; pulse 120, very feeble; respirations 28. She was noisy and rambling last night, and is quite incoherent this morning—refuses food; bowels torpid; tongue swollen and glazed. Acute delirious condition has so far subsided as to permit her partially to understand what is said, and to reply coherently; compulsory feeding has still to be resorted to." The following day it is stated that—"Patient was more than usually excited again last night, repeatedly sprang out of bed, and jumped into the patients' beds. Slept one hour after two ounces of stimulant. She remained sleepless and wild all night, despite a sedative then given. Has taken her food for the first time voluntarily."

On the fifth day following her admission, she was fairly calm and rational, having slept some four hours during the night; but there was now noticed a considerable swelling over the left parotid region, so that she could hardly open her mouth; the lobe of the ear was also red and inflamed, the pulse had improved in quality. From this date, the patient improved rapidly in mind, and she was *quite convalescent* three days after the appearance of the swelling. The latter had extended over the mastoid region and down the neck, quite obscuring the angle of the jaw; the integument is of a rather congested redness, thickened, and the swelling hard and tense; the left eye is completely closed by great œdema of the lids; temperature has fallen from 102° to 100°, pulse 108. There is considerable tumefaction of the left tonsil. Suppuration occurred in the swollen part, and discharge took place from the external meatus three days later. No relapse of mental symptoms occurred, and patient left in six weeks from the date of her admission.

J. G., a married man, aged forty-nine, by occupation a plumber, had been treated at home for the past month for mental symptoms of a maniacal type; he had violently assaulted his wife and threatened her life. His mental disturbance was attributed by his friends to excessive drinking; one point was certain—he had no insane or neurotic heritage, both parents had lived healthily to a good old age, and no other member of the family had been mentally affected or had suffered from nervous disease. His drinking habits had extended over a period of many years; and evidence of nervous disease or mental flaw had undoubtedly been regarded by his friends as but the result of intemperance. Probably he had been deranged for much longer than was stated; yet he had worked at his regular occupation up to a few weeks of admission. When admitted, he was at once recognised to be the subject of general paralysis; he had pin-hole pupils (spastic myosis); his voice and lips were tremulous; he had suffered during his journey to the asylum what the Relieving-officer believed to be a "stroke."

But the important feature about his state was the intensity of his maniacal reductions; he was evidently in a profoundly prostrate condition, and was likely to sink rapidly from acute maniacal delirium. His urine was retained, and had to be withdrawn by a very small catheter, owing to his having a contracted prepuce with extremely minute aperture; surgical measures, however, were at once adopted to relieve this state. He could not stand upon his feet, but immediately "doubled up," and lay for the most part in a helpless, prostrate, dorsal decubitus. Acute visual hallucinations were constantly present; he made continuous snatches with his hands as though to grasp imaginary objects, and lay muttering utterly incoherent gibberish. There were much tremor of the limbs, and muscular jerkings generally. Patient's consciousness was so far obscured that he failed to appreciate the purport of anything said or done for him. Paraldehyde (mins. xxx.) was administered, but wholly failed to induce sleep; strong nourishment of milk with eggs, essence of beef, and concentrated foods, was given him, but with much difficulty, owing to his resolute resistance and terrified state of mind. He was pale, pinched, and haggard, and continually restless through the next day, requiring regular catheterism, a normal amount of urine being each time withdrawn. The following night he obtained no sleep, tossing about restlessly, and muttering incessantly; the heart's action was becoming excessively enfeebled, his limbs cold, and his lips slightly cyanosed. Every precaution was observed, and small quantities of nourishment were given frequently to keep his body warm and stimulate the circulation; but he died the following day from cardiac failure.

FULMINATING PSYCHOSES.

Contents.—Uniform and Partial Denudations—Defective Control—The Neurotic and Criminal Subject—Nature of Impulsive Insanity—Insane Homicidal Impulse—Existence of Aura—Epigastric Aura—Uncovering of the Brute Instincts—Relief of Mental Tension—Illustrative Cases—Suicide in Homicidal Subjects—Etiology—Effect of Physiological Cycles—Epilepsy—Masked Epilepsy—Alcohol and Impulsive Insanity—The Mimetic Tendency—Suicidal Impulse—Kleptomania—Dipsomania—Erotomania—Imperative Obsessions.

The dissolutions of the nervous system which issue in insanity by no means reduce the subject to pre-existent levels of mental life corresponding in all respects to former stages of evolution; the **denudation,** to use an apt term, is by no means so **uniform** that the mental wave recedes along the whole line of its former advance. Such a uniform recession does occur in physiological senescence, and is still more pronounced in the premature decay of senile dementia; but, in most forms of insanity, the denudation is a **localised** one, or, at all events, begins in many separate areas, and the resulting mental disturbance is wholly unlike any of the results of a uniform physiological denudation. The general results also will vary with the intensity and rapidity of the diseased process, and the factors so often insisted upon by Dr. Hughlings-Jackson in his studies of convulsive diseases must also not be neglected in considering the less acute processes of mental disease.

It is by these **partial denudations** that we seek to explain the incongruous results of the diseased process and the overbalance of faculties so characteristic of mental disease. At no stage in the history of insanity, except, perhaps, the senile forms, do we find the man altogether reduced to the mental state of childhood—a *plus* or *minus* quantity ever prevents an exact parallel being drawn, so that we readily distinguish such anomalous reductions from the results of a uniform physiological or pathological denudation. Certain features which characterise the mental life of the child spring into obvious prominence in the adult subject of mental disease. The infantile mind is above all things characterised by the lack of *control*—its instincts, passions, desires, actions, all alike, exhibit in a high degree a want of inhibitory restriction, and the further development through childhood and youth to adolescence and adult age is a record of the slow progressive superposition of controlling centres. Normal mental development is specially characterised by this uniform and progressive establishment of self-control (so to speak) upon higher and still higher levels; but, just as we get in the *dissolutions* of disease *partial* denudations—so here, in the progress of mental *evolution*, we meet with developmental phases of a monstrous character, presenting, not the normal uniformity of level, but the bizarre irregularities, exaggerated here and defective there, which signalise so frequently the neurotic heritage of the subject. Defective control over certain animal passions and instinctive desires (often associated with an intense staccato restriction over others, amounting to a morbid hyper-sensitiveness) is a peculiar characteristic of such predisposed subjects; whilst a still more universal defect of the inhibitory faculty is illustrated by the criminal class of the community. The reductions of mental disease, therefore, will more readily find their parallel in the various anomalous developmental phases of the neurotic subject, or in the extremes of inhibitory defect presented by the criminal, rather than in earlier stages of the healthy and normally-developing brain, and our studies of these developmental types should facilitate our comprehension of the varied reductions of insanity.

Much may be said of the ill effects of injudicious training of the mental faculties of the young ere they have attained an age when such faculties should be called into operation; and we quite agree with Dr. Clouston that different brains attain their power of control at different ages, and we also have seen "many children whose anxious parents had made them morally hyperæsthetic at early ages through an ethical forcing-house treatment"; but we opine that all pronounced instances of the kind are *neurotic subjects*, as in the case of the little boy of four mentioned by him, "who was so sensitive as to right and wrong, that he never ate an apple without first considering the ethics of the question as to whether he should eat it or not"—yet who was, at the age of ten, "the greatest imp I ever saw, and could not be made to see that smashing his mother's watch, or throwing a cat out of the window, or taking what was not his own, were wrong at all."* What we specially insist upon here is the fact that the subject presenting such mental distortions is not the product of a vicious educational code so much as the victim of an organised neurotic heredity; and that we should in these developmental forms learn to recognise features common to them and the reductions of mental disease. So also as regards the true criminal type, the difficulty of drawing any clear line of demarcation between crime and insanity is well recognised; certain forms of insanity, more especially the so-called "moral insanity," presenting peculiar difficulties to our arriving at a conclusion as to the degree of criminal responsibility involved in the case. Nor need this fact surprise us, since the one presents us with partial developmental arrests at levels to which the brain of the insane must frequently become reduced; what must always be kept in mind is the fact that the one is the outcome of a developmental failure, or vice, the other is a genuine dissolution.

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* *Op. cit.*, p. 311.

All acute forms of insanity are peculiarly characterised by this loss of control. We recognise it in the failure of attention and the incoherent flow of ideas expressed in rambling speech, in the unrestrained passions, varying moods, incessant movement, gesture, and all the outrageous conduct of the maniac; but it is not in these universal and complex disturbances of faculties that we find the symptoms of "impulsive insanity," as generally understood by that term. There are mental affections in which the chief, nay, the sole discoverable feature is this failure of inhibition exhibited in ungovernable, sudden impulse, and in entirely unrestrained conduct, whilst the intellectual and emotional spheres remain wholly or only in part unaffected. It is in this freedom of the affective sphere of mind from implication, and the purely impulsive nature of the act, that we must learn to recognise the genuine impulsive insanity, as understood by older writers. Both Pinel and Esquirol at first doubted the existence of pure insane impulse apart from intellectual flaw or delusion; and many authorities of repute have since their day considered the doctrine a dangerous as well as a fallacious one; yet eventually Pinel and Esquirol asserted the existence of this terrible malady, and painted its distinctive features in no uncertain colours. Either there is, or there is not, such a disease as impulsive insanity; and we must remember that our denial of its existence carries with it the implication that the impulsive conditions which we recognise in a minor degree in healthy physiological states, such as the almost irrepressible desire to break a delicate glass globe held in the hand, and many other similar experiences which we are all familiar with, cannot arise in an absolutely uncontrollable form as the result of pathological disturbance. It may appear to the student an *unnecessary refinement* to insist upon this distinction, but a moment's consideration will assure him that the distinction is one of vital import, not from its scientific bearing only, but more especially from its medico-legal aspects.

The lawyer is naturally suspicious of the existence of this form of insanity, and is, very properly, guarded in his acceptance of the doctrine which carries with it such far-reaching results; he perceives the difficulty of distinguishing between what is and is not controllable —between an insane impulse and the outcome of criminal volition; and he, moreover, perceives the difficulty—nay, the impossibility—of recognising its existence, and at the same time reconciling it with the legal criterion of responsibility: and, lastly, he must recognise that the admission of this fact throws on the medical witness the full responsibility of defining what is and what is not of the nature of an incontrollable impulse.

Great as may be the difficulty in many cases, of clearly distinguishing between the blind incontrollable impulse of the insane, and the rash, impetuous act of the responsible criminal, we must not shrink from the imperative duty of affirming the existence of this form of insanity if our clinical experience justifies the belief, so momentous are the consequences embraced by its acceptance or rejection.

In insisting upon such a distinction, we must not forget that it is more or less an arbitrary one—that nature imposes no such absolute line of demarcation between what we elect and what we do not elect to the dignity of morbid types; that in reality, one, or a few, or many of the mental faculties may be deranged, and in all possible degrees of intensity, and so forms of impulsive insanity may merge into forms characterised by intellectual or emotional disturbance; and *vice versâ*, intellectual impairment with delusion may merge into the typical forms of impulsive insanity, exhibiting every shade of transition from the one to the other type. What is of still further import is the fact—which clinical experience very strongly emphasises—that alternations of pure *impulsive* insanity and forms of *intellectual* or *moral* insanity occur in many insane subjects.

Nature of the Insane Homicidal Impulse.—In the first place, we should note the **causeless** or **motiveless** nature of the act; the impulses arise wholly apart from any incentive, delusional or otherwise, nor is the victim able, in the great majority of cases, to trace any connection between any pre-existing emotional or intellectual phase, and the onset of the insane impulse. Its irrelevancy to surrounding circumstances is in itself so characteristic a feature, that the subject invariably insists strongly upon this fact. Suddenly, amidst, it may be, the pleasures of the family circle, or at the moment of devotional exercise, to the intense horror of the subject, the morbid feeling suggests itself without any obvious provocation (like a phantom demon), and requires all his efforts to dispel it. The horror of the position will often drive the sufferer to a free confession of his state, and to urgent entreaties for protection against such unbidden mysterious impulses, as numerous cases attest; but instances occur, where the unfortunate subject has struggled for years with his infirmity, and never revealed his deadly secret until compelled to do so upon the commission of some desperate act.* The motiveless nature of such acts may be called in question, and grave suspicion be expressed, from the admitted difficulty of always assigning a consistent motive even for the acts of the sane; but, just as readily as we may err in imputing no motive to an act when such is not clearly obvious, so, we may even more easily fall into the opposite error of assigning a wrong motive to an insane impulse, influenced by accidental circumstances in which the subject happens to be placed. The motiveless nature of the morbid impulse is forcibly illustrated by cases in which the subject suffers from such feelings when no one is near, or at the moment of awaking from sleep; for, as in the case of the suicidal impulse (an impulse which is equally transient), the commission of the fatal act is often averted by the absence of opportune means; so, in the homicidal impulse, the morbid energy is dissipated and the murderous act averted by the absence of the object.

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* See, on this point, Drs. Bucknill and Tuke, *op. cit.*, p. 268.

In the second place, we must note the prodromal indications and accompaniments of the insane impulse so far as they are afforded by subjective and objective indications. The subjective accompaniments vary considerably in different cases; in many, the cerebral discharge which initiates the impulse, is productive of a **genuine aura** such as often precedes the epileptic convulsion. The morbid sensation is often *peripherally* referred, is of sudden accession, and may rapidly pervade distant parts of the body. Thus in the case of one unfortunate victim (Reg. *v.* Mountain), we are informed that an intense burning heat suddenly seized him in the epigastrium and was rapidly transferred to the throat, accompanied by a sense of constriction and urgent thirst, upon which the homicidal fury arose, and momentarily bereft him of all control.* Others complain of colicky pains, a sense of heat in the abdomen or chest, headache, restlessness, loss of appetite, and lowness of spirits (*Taylor* †), of sensations referrible to the head, "flushings of blood to the brain," a sense of constriction or tightening, as of a ligature, round the scalp, or of a feeling as if a cold fluid were poured upon the head and along the spine. Dr. Skae, in his Annual Reports for 1866 and 1868, describes a well-marked aura as preceding homicidal impulse. In certain other cases a definite hallucination of the special senses may be the immediate forerunner of the homicidal impulse. The connection of these phenomena with epileptic discharges is often apparent in such subjects, and the following case, quoted by Maudsley from Marc, seems to indicate the repression of the impulse by the arresting of the aura:—

> "Mr. R., a distinguished chemist and poet, of a naturally mild and sociable disposition, placed himself under restraint in one of the *maisons de santé* of the Faubourg St. Antoine. Tormented with an homicidal impulse, he prostrated himself at the foot of the altar, and implored the divine assistance to deliver him from the atrocious propensity, of the cause of which he could give no account. When he felt himself likely to yield to the violence of it, he hastened to the head of the establishment, and requested him to tie his thumbs together with a ribbon. This slight ligature was sufficient to calm the unhappy R., who subsequently endeavoured to kill one of his friends, and finally perished in a fit of maniacal fury."

The epigastric aura, followed by spasm of the throat and intense thirst, alluded to above in one case, has been noted in other subjects;[*] it is of interest as indicating a primary disturbance of the vagus, and as giving rise to the most intense and massive feelings of organic life, which in the brute arouse the most ferocious instincts.

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* See in this connection an instructive case of homicidal and suicidal insanity recorded by Dr. Frank A. Elkins, *Edin. Med. Journ.*, 1890.

† *Med. Jurisprudence*, vol. ii., p. 553.

Thus, Professor Bain says—"They (the feelings of inanition and thirst) are far more intense than mere nervous depression, and, therefore, stimulate a more vehement expression and a more energetic activity. Even when not rousing up the terror of death, they excite lively and furious passions. The unsophisticated brute is the best instance of their power." And again, "There is something intensely kindling in the appetite of carnivora for food, which rises to fury when the flesh is scented out and begins to be tasted."[†]

The association of these organic sensations with the springing into life of the brute propensities in the human subject is, we take it, a suggestive fact. Certain objective indications of the morbid process are also occasionally afforded, chiefly of vaso-motor origin—intense pallor may precede the act, or the face may become suffused, and a copious sweat break out over the body as the impulse is resisted and subsides; the heart usually shows excited action, and the arteries of the neck and temples pulsate violently.

In connection with outbursts of homicidal frenzy the Amok of the Malays affords instructive parallels amongst lower states of civilisation. Suicide amongst the Malays, it appears, is extremely rare (*Ellis*), but the paroxysms of murderous frenzy known as "running amuck," appear clearly to be associated with a sub-conscious automatic mental state, as in genuine epileptic and alcoholic automatism, the agent being left wholly oblivious to his murderous deeds. "Amoks result from an idiosyncrasy or peculiar temperament common amongst Malays, a temperament which all who have had intercourse with them must have observed, although they cannot account for or thoroughly understand it. It consists in a proneness to chronic disease of feeling, resulting from want of moral elasticity, which leaves the mind a prey to the pain of grief, until it is filled with a malignant gloom and despair, and the whole horizon of existence is overcast with blackness. . . . These cases require discrimination on the part of the medical jurist to prevent irresponsible persons suffering the penalty of the injured law." (*Dr. Oxley*, quoted by Gilmore Ellis.)

The intensity of the morbid process is further indicated in the utter loss of self-control. "Everything passes out of mind," said one such unfortunate subject to us, "except the one thing I wish to accomplish —I can think of nothing but the desire to kill some one." The one burning idea prevails to the exclusion of all others at the height of the attack, and is (in all respects, as Maudsley has insisted) "a convulsive idea springing from a morbid condition of nerve-element, and comparable with a convulsive movement." * In other respects the condition shows its kinship to the convulsive neuroses, viz., in the immediate relief afforded by the accomplishment of the act, or the dissipation of the morbid energy in other directions; like all transient nervous discharges from the cortex, the associated mental tension is instantly relieved thereby. Yet, it must not be forgotten, that the impulse is in many instances successfully resisted; and that the early history of many cases of homicidal mania is one of a long-continued and secret struggle of the victim against the morbid feelings which create in his mind a dread and a horror indescribable. Fully recognising the atrocious nature of the crime to which he seems impelled, he is in constant dread lest in some weak moment his power of resistance, already enslaved, should wholly succumb in the frenzy of the seizure. Instances have occurred where this struggle was carried on for years—thus in the case of Reg. *v.* Mountain the prisoner admitted the existence of such insane impulses for a period of ten years prior to the murder, the exceptional atrocity of which, with other related circumstances, make it worthy of note here.

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[*] See a case by Pinel quoted in Dr. Maudsley's *Responsibility in Mental Disease*, p. 141.

[†] *The Senses and the Intellect*, Alex. Bain, pp. 126 and 253.

The prisoner was a young man, aged thirty-two years, of undoubted neurotic heritage; his mother, maternal grandmother, and maternal aunt had been insane; his maternal uncle had cut his throat, and his brother was of feeble intellect. The maternal aunt, who was under our observation for years, was the subject of suicidal impulses, had tried on several occasions to strangle, to hang, or to drown herself, as the result of imperative feelings distinctly arising from the group of organic sensations. The unfortunate subject of such ancestral frailty had always been timid and unnaturally suspicious; but no decided delusional phase had been observed at any time until a few months preceding the murder, and then only as the immediate outcome of drink. Ten years prior to the event in question, he first became subject to the peculiar sensations which we have already referred to, and which were invariably the forerunner of intense homicidal impulses; they almost invariably occurred at times when he was alone, and he would pace wildly up and down his room to "work the feeling down;" and often he has rushed from the house where his aged mother and servant lived, when he felt the feeling arising, lest he should not be able to resist the murderous impulse. He had struggled against these feelings, and "prayed to be delivered from them" in agony of mind without success for years. They were increasing in intensity, and to add to his misery his natural nervousness and suspicion were also more prominent.

His mother's attack of insanity some years previously, and his aunt's state (when he had visited at the asylum), constantly preyed upon his mind, and engendered the feeling that he would become insane. He had kept his dangerous feelings and propensities a profound secret, so that his closest acquaintances had failed to recognise any indications of his real condition until, latterly, when he gave way to drinking; and then it was observed that very small quantities of alcohol produced grave mental disturbance, characterised by persistent delusions of persecution and errors of identity. On two occasions he suffered from genuine attacks of maniacal excitement, but of transient duration only. As a natural result of these intemperate habits, his former symptoms became further intensified; yet, up to the evening of the murder (except when under the influence of drink), no intellectual disturbance was recognised by his friends. On this evening, after taking stimulants freely, he locked his mother and the servant girl in a room together, and in the most brutal and atrocious manner attacked his mother, kicking her to death, and causing the most horrible mutilation of the body, keeping the girl at arm's length by a loaded pistol. For five hours this brutal violence was continued, he meanwhile affording abundant evidence of a deluded state of mind by his conduct and utterances. His subsequent condition upon arrest was consistent with an attack of mania-a-potu. Subsequent to his recovery from the alcoholic delirium, he had experienced a return of homicidal impulses in prison, a man who slept in the same room having nearly been a victim to his murderous frenzy. The prisoner was considered irresponsible at the time of the murder on the ground of insanity, and was ordered to be retained during Her Majesty's pleasure.

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* *Op. cit.*, p. 156.

The case is of interest in its medical aspects as reproducing some of the most important features of homicidal impulse in the insane. There is the fact of the peculiarly hereditary nature of impulsive insanity; there is the strange association of deranged organic sensations with the convulsive conduct; there is the emphatic proof of the fatal effects of alcoholic indulgence in such cases, and the ready passage into delusional forms of insanity; and, lastly, there is the secrecy so often maintained by the subjects of this form of malady, lasting over a period of ten years. See a notable case recorded by Dr. Tuke.*

We have little doubt that many instances of mysterious suicides are to be accounted for by the prevalence of homicidal feelings—the victim tortured by the terrible secret seeks relief in self-destruction rather than reveal his condition, or subject those near and dear to him to any further risk. The condition of the homicidal subject immediately subsequent to the act is characteristic—it is usually one of complete relief from anxiety, and utter indifference to the enormity of his crime; frequently his first act is to coolly confess his crime and give himself up to justice.

Exceptions, however, occur where the subject of insane impulse endeavours to conceal his crime like the responsible criminal. Thus, at the West Riding Asylum, a subject of such impulses secured an iron bar and struck a harmless imbecile patient on the head as he lay asleep within a few yards, fracturing his skull seriously, and then deliberately concealed the instrument in some shrubbery near at hand, and coolly took up the paper he had been reading a moment before, apparently free from the least concern. Up to the present day, four years since his homicidal act, he denies positively any knowledge of the affair, and he exhibited the utmost indifference on being questioned immediately subsequent to his violence. In fact, his nonchalance at the time, and his subsequent behaviour, might almost have been regarded as consistent with the impulsive automatic act of an epileptic, were it not that the subsequent history of the case revealed clearly the existence of insane impulses preceded by a definite aura, but not of genuine epileptic paroxysms; and, moreover, proved him to be possessed of considerable insane cunning. In the genuine impulsive forms of insanity, consciousness is never so far impaired as to issue in forgetfulness of the details of the homicidal act. When such is the case—when any marked obscuration of memory is apparent—we may presume the impulse to have been of epileptic origin, or to be the outcome of alcoholic delirium.

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* *Psychological Medicine*, 1874, p. 268.

Etiology.—In all these cases of pure impulsive insanity there is, we believe, a well-established basis of a neurotic heritage, and if the individual's history is scrutinised with sufficient care, we are assured that evidence of mental instability will be discoverable throughout his life. It is, however, at the critical epochs of life that this predisposition especially tends to assert itself—periods at which grave nutritional disturbances are prone to arise in the central nervous system, inducing the peculiarly convulsive outflow of nervous energy which characterises these epochs, even in normal physiological operations. *Puberty* and the *climacteric* are prone to the convulsive type of the neuroses, and the same prevails at the *puerperal period* during *lactation*, and associated with the various forms of *menstrual derangement*. Several instances are on record where the revolutionary epoch of puberty has aroused the homicidal feelings in youth, as in the case of Margaret Messenger, aged thirteen years, who killed her brother and drowned another child, six months old, without any discoverable motive. Young girls suffering from temporary menstrual derangement are subject, as is well-known, to various perverted instincts and appetites, and the hysterical outbursts are often associated with an almost irresistible tendency to destructiveness, and not very rarely with a homicidal feeling (see case of Reg. *v.* Brixey).* We have known several instances where the subject has expressed her dread of sleeping in the same room with other members of the family, and of being left alone with her younger sisters, lest she should not be able to restrain the impulse felt to injure them.

The **climacteric** in woman is a period during which mental disturbances are frequently associated with suicidal impulse; but, as we shall see later on, the impulse is usually the outcome of intellectual derangement and grave delusional perversions. Yet, homicide and suicide may occur at this epoch as the result of a purely impulsive condition, and more particularly in such cases as have developed intemperate habits. The **puerperal period,** as is well known, renders neurotic subjects liable to insane impulse, and, although usually a symptom of the general disturbance of puerperal mania, the simple instinctive form may alone prevail. We recognise a similar condition in animals, which, in the deranged states following parturition, will kill and even devour their young. Epilepsy is a frequent source of these depraved and resistless feelings. Homicidal impulses may prevail in one of four conditions in the epileptic subject, viz. :—

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* Quoted in Taylor's *Medical Jurisprudence*, vol. ii., p. 564.

(*a*) In epileptic furor or mania, associated with hallucination or delusion;

(*b*) In the so-called "epilepsia larvata" (*Morel*), the "masked epilepsy" of Esquirol;

(*c*) In the dreamy state of epilepsy; or, lastly,

(*d*) As a simple impulsive derangement during the inter-paroxysmal period.

It is the latter alone which can be regarded as genuine Impulsive Insanity; the three former conditions are attended by such general mental derangement as to exclude them from the category of pure affective forms of insanity. It will be more convenient, however, to refer briefly to such forms at the present juncture, and to deal with them in further detail in our remarks upon insanity associated with Epilepsy. In the reductions of Epileptic Mania, or the post-paroxysmal excitement, of which we see so much in our asylums, the homicidal impulse springs into life almost invariably as the result of delusion. The murderous act is traced to a pre-existing delusional state, with which it has often a direct connection (see case of Reg. *v.* Taylor, see *Alcoholic Insanity*); or, again, hallucination of sight or hearing may prompt the act—a voice may be heard commanding the epileptic subject to kill, and the impulse arises in resistless force (case of *E.C.*, see *Epileptic Insanity*)—or a visual hallucination, in the form of some object of terror, may call forth these same results. The epileptic furor may be of some considerable duration, and the subject remain in a dangerously homicidal state during its continuance; but subsequent to the paroxysm, the subject will remain either greatly bewildered (retaining only very partially some fragmentary recollections of the attack), or, still more commonly, be wholly oblivious of the circumstances and of the conduct which he has just displayed.

In the **masked epilepsy** of older writers, we find that a fit of homicidal mania may replace the convulsive seizure (a convulsive idea, as Maudsley would say, takes possession of the mind), and, without any of the usual epileptic phenomena preceding, a sudden irresistible murderous impulse (probably prompted by delusion or hallucination) occurs; but here, again, the subject fails to recall any conception of his actions. So, likewise, in the dreamy state of epileptics, approaching the somnambulistic condition, homicidal acts have been committed in a semi-unconscious automatic state of mind. It is astonishing how complicated may be the acts performed in these states by the epileptic automaton. A better illustration could not be found than that quoted by Dr. Gowers, where a carman in this state of automatism, after an epileptic seizure, "drove through the most crowded parts of London without any object, but also without any accident."*

The Amok of the Malays already referred to (p. 210) is attributed, most plausibly, by Gilmore Ellis to **masked epilepsy**—alcoholic reductions being wholly out of question in view of the great aversion of the whole race to alcohol.†

Genuine impulsive insanity, apart from grave mental derangement, however, is also occasionally seen in epileptic insanity; in the intervals between the convulsive seizures, certain patients are subject to frequent insane impulses to murder (without any motive or malice) any one with whom they are brought in contact. These conditions usually alternate with delusional states, and with the maniacal outbursts succeeding the epileptic attack—they are the most anxious cases to treat, and the most difficult patients to control. Such subjects are peculiarly susceptible to the effects of small quantities of alcohol, which may induce, even in very trivial amount, the most furious outbreak of mania, or the impulsive homicidal state alluded to.

Alcoholic excess may induce the impulsive form of insanity in certain predisposed neurotic individuals; a condition of alcoholic delirium of extremely short duration (mania transitoria)‡, in which a mad impulse to murder prevails, may thus be induced by what is usually considered by no means immoderate drinking. The symptoms, however, embrace much mental confusion, and the subject remains, after the attack is over, in a state very similar to an epileptic after an attack of petit mal.§ Amongst other etiological factors we must not fail to note the vicious agency of *imitation* which was originally emphasised by Esquirol, as one of the causes of this affection. Undoubtedly, the morbid excitement engendered by the perusal of records of criminal horrors, by the publicity afforded in our Assize Courts to the revolting details of crime, and, up to within the last few years, the demoralising effect of public executions have greatly fostered the development of these states of mental disease. If there is one fact in mental physiology more established than others, it is that the continuous direction of the mind to the sensual and purely animal passions of our nature tends to intensify their potency—to render their channels of operation more pervious, and so to withdraw them from the inhibitory control to which they should ever be subject. The brutal instincts are still less protected in those persons of weak mind, who, not endowed with an average amount of controlling power, require but the intensification of such instinctive states to lead to explosive outbursts; in such cases mental strain, anxiety, ill-health, and other exhausting conditions, and especially alcoholic and sexual intemperance, may readily lead to attacks of homicidal mania at periods when the public mind is horrified by some startling crime.

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* *Diseases of the Nervous System*, vol. ii., p. 691.

† See a most instructive article with cases on "The Amok of the Malays," by W. Gilmore Ellis, M.D., Singapore, *Journ. of Mental Science*, July, 1893.

‡ Maudsley is undoubtedly correct in asserting that many cases of *mania transitoria* are really instances of "mental epilepsy."—*Op. cit.*, p. 230.

§ On the subject of mania transitoria Maudsley remarks :—Although epilepsy, masked or overt, will, I think, be found to be at the bottom of most cases of mania transitoria, it must be admitted that there are some cases in which there is no evidence of epilepsy in any of its forms to be found; but it may well be doubted whether a distinct insane neurosis is not always present in these cases. With such a constitutional predisposition, a genuine attack of acute insanity, lasting for a few hours only, or for a few days, may break out on the occasion of a suitable exciting cause, and during the paroxysm homicidal or other violence may be perpetrated. After childbirth it sometimes happens that a woman is seized with a paroxysm of acute mania of short duration, during which perhaps she kills her child without knowing what she is doing. The effect of alcoholic intemperance upon a person strongly predisposed to insanity, or upon one whom a former attack

The Suicidal Impulse.—What we have said respecting the homicidal impulse applies in most particulars to the self-destructive propensity; it also arises in subjects who exhibit no intellectual disturbance, and in whom the moral sense is intact, in so far, that they, recognising the horror of their situation, and the unnatural character of the morbid promptings, revolt against the perpetration of the act. Like the homicide, they may implore protection, and voluntarily resign themselves to asylum supervision, dreading lest they may be overmastered by the suicidal impulse. So likewise do we find the impulse of convulsive nature sudden in its onset, transient in its course, and followed by immediate and complete relief; its analogy to the epileptic state being still further indicated by the occurrence of an aura, usually an aural hallucination. The condition to which we allude is, of course, not the ordinary suicidal tendency of simple melancholia, where the morbid depression precedes and explains the negative suicidal state; but the condition where, from the first, the suicidal propensity presents itself, any depression being secondary, and induced by the patient's helpless condition. The climacteric epoch not unfrequently develops this impulsive form of insanity just as it does homicidal states; and a good illustrative case is detailed further on in our study of the insanity prevailing at this

has left predisposed to a second, is sometimes a short but acute mania of violent character with vivid hallucinations and destructive tendencies; and a like effect may be produced by powerful moral causes, sexual excitement, and other recognised causes of insanity." (*Responsibility in Mental Disease*, p. 247.)

period of life (see case of *S. H.*). Winslow records the statement of one patient as follows:—"For six months I have never had the idea of suicide, night or day, out of my mind. Wherever I go, an unseen demon pursues me, impelling me to self-destruction. My wife, friends, and children observe my listlessness and perceive my despondency, but they know nothing of the worm that is gnawing within."*

The morbid impulses to which the insane are prone are as diverse as are the active tendencies of the mind itself; but many manifestations of an instinctive character, on account of their obtrusive and criminal nature, have claimed special attention, and have been exalted by some authorities into distinct forms of insanity; whereas, in truth, they should be merely regarded as symptoms or a group of symptoms **(syndromes)** which may occur in *any form* of insanity, and which, in particular, characterise the mental alienations of the degenerate. Such are cases of **kleptomania, pyromania, dipsomania**, and **erotomania,** or the impulse towards theft, incendiarism, drunkenness, and sexual outrage. A few remarks on these so-called **obsessions** may deserve a place here.

Kleptomania.—The essential character of this condition is, like that of all morbid, instinctive acts, more or less incontrollable, despite any resistance to its perpetration the subject may exert. It is not a mere propensity towards thievish acts, such as characterise so many forms of insanity; but, the idea of possessing what is not rightfully theirs, attended often by great anxiety and terror lest control be lost, becomes in itself the originating factor which determines the theft. The painful feeling preceding the crime vividly portrays the lack of control and the cruel helplessness of the victim, the *intensity* of the idea being the immediate precursor of the impulsive act. In general paralysis, the early stages are often characterised by thievish propensities, but the nature of the act is wholly different; the stupidity and awkwardness of the deed are in themselves sufficiently indicative of its being the outcome of **dementia,** just as are the other moral lapses which appear in this disease (p. 287). In other forms of dementia and imbecility, and in the confusional reductions of epileptic insanity, the thievish act can usually be at once distinguished from the impulses of the kleptomaniac; yet, in epilepsy the genuine condition may also be revealed. In the latter case, it may be an indication of transient reduction, in which, higher controlling centres being in abeyance, the morbid propensity springs irresistibly into activity; or it may be the outcome of *permanent moral lapse* from the continued progressive denudation of this disease.

What is all important for us to realise is the fact that just as by diseased processes (epilepsy), by toxic agencies (alcohol), or again, by functional excesses (sexual), the volitional control is so paralysed, as to admit of over-action throughout the whole gamut of morbid propensities, so also in the degenerate organism, where inhibitory centres have always been of rudimentary development, any of these pseudo-moral lapses may betray themselves as genuine convulsive or impulsive acts. Kleptomania has been affirmed to be a frequent accompaniment of menstrual derangement and the late pregnant state.

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* *Obscure Diseases of the Brain*, p. 265.

Dipsomania.—The same features pertain to this condition; there is an all-powerful, irresistible tendency to drink, which appears after a more or less mental depression, vague sense of impending evil, and a general functional disturbance; it shows a paroxysmal or intermittent nature, and the debauch, often leading to the greatest excesses in other directions, is often followed by a keen sense of shame and penitence, and also by an utter indifference or distaste for stimulants in the interval. Its rhythmic or paroxysmal recurrence appears to indicate nutritional anomalies as the basis for the morbid syndrome, and, in most cases, there is undoubted evidence forthcoming of a neurotic taint. In all cases the phenomena are alike; first, the obsession or idea of drink; then the anguish aroused by the sense of volitional impotence; and lastly, the incontrollable impulse to drink, leading often to the most immoral, degrading, and vicious conduct to secure the intoxicant. In the facts that the taste for drink does not *first occur*, that, indeed, the victim often does not care for drink for its own sake, and that alcoholism does not usually lead to this disease, we at once perceive the distinction from ordinary cases of drunkenness. The essential features are, in short, the impotence of will before a cruel obsession, the short but painful struggle to resist, the impulsive outbreak, a morbid syndrome occurring in a degenerate subject whose inheritance has probably revealed itself throughout life in mental instability, in general want of balance, and in the features which notably present themselves in all forms of the explosive psychoses.

Erotomania.—The same remarks apply almost in every particular to conditions of erotomania, which embraces so-called "satyriasis" and "nymphomania," terms applied to the erotic impulses occurring respectively in the male and female subject. Satyriasis appears occasionally *after* epileptic discharges, claiming on this account special attention at the hands of the medico-jurist. Instances of perverted sexuality, again, often include cases of true impulsive insanity, but the whole subject has received such special attention from Continental writers that the reader may be referred to the voluminous works of Lombroso, Krafft-Ebing, Caspar, and others for further particulars.

Obsessions, Imperative or Dominant Ideas.—When any one idea or group of ideas invade the mind *automatically* to the exclusion of others, despite the effort of the will to suppress or eliminate the same, we speak of it as a dominant or imperative idea—an obsession. Such obsession may alone present itself, as an isolated fact, but it is usually the initial stage preceding irresistible *impulse*.

The effective capacity of the will rises and falls with the resistance presented to it—*e.g.*, with the vividness of the idea to be suppressed; and we are well acquainted with conditions where the morbid vividness of the idea is such that the will is powerless to inhibit, although exercising its normal energy. In such a case, we cannot speak of the morbid syndrome of obsession and impulse as due to enfeeblement or disease of the will, but rather to an overpowering morbid tendency wholly disproportionate to the normal volitional activities of the subject. Obsession, or imperative ideas become, in this sense, a sort of mental monstrosity, and must be clearly distinguished from such phenomena as epileptic impulse, where genuine reductions have removed volitional control, with the result that instinctive actions spring instantly into life.

Such painful obsessions are usually found in the subjects of a neurotic heritage—ancestral epilepsy, alcoholism, insanity or crime being most frequently traced. The degenerate offspring of a neuropathic stock becomes still more subject to such obsessions by agencies which tend to reduce the volitional control—alcoholic and sexual excesses in particular. The frequent and persistent direction of the attention to certain morbid ideas will also tend in the degenerate to establish such obsessions, and favour their relief by the corresponding impulsive act. There is little doubt but that the morbid and repulsive sensationalism of our Assize Courts, thronged as they are by unstable and degenerate individuals, largely fosters the tendency to the development of obsessions and the fulminating psychoses, through misdirected imagination.

Amongst those most generally met with are the obsessions of fear and indecision. The fear may pertain to any person, place, object or relationship; the category of the things feared being limited only by the possibilities of existence. This morbid dread, however, pertains nearly uniformly to some definite object, and the innumerable terms invented to designate these mental states illustrate forcibly the absurdity of attempting the symptomatological classification of such mental manifestations. A fear of pollution by places and things; a fear of closed or open spaces; a dread of certain animals; of the sight of blood, knives, poisons, explosives—are some of the more common, but the list might be almost indefinitely extended.

The obsession of doubt again resembles that of fear, having as its basis an utter distrust of self; *e.g.*, there may be a tendency to review incessantly over and over again one's own actions; to repeat again and again numerical calculations; to interpose repeatedly an incongruous and wholly irrelevant word or phrase in the course of conversation; or to enumerate over and over objects lying before his gaze. One notable case known to the writer would present herself a dozen times a day at a railway station, but never could persuade herself to undertake the short journey intended; in this case complete recovery was secured.

Closely allied to the foregoing are the states where there is inability to rise, to walk, or to sit down (astasia, abasia), and which have been closely studied by Continental writers in hysteric subjects. In these conditions, the will does not fail in its inhibitory capacity, as in restraining a morbid impulse, but it fails to initiate the action desired; such states are known as "aboulias," or "aboulic obsessions."

STATES OF MENTAL ENFEEBLEMENT.

Contents.—Mental Deprivation in Contradistinction to Developmental Arrest—Persistent Enfeeblement—Chronic Residue of Asylum Communities—Recoverability of Maniacal and Melancholic Forms—Consecutive Dementia—Delusional Insanity—Genesis of Monomaniacal States—Environmental Resistance—Transformation Completed—Mystic Symbolism—Illustrative Cases of Delusional Insanity—Monomania of Pride (J.O., E.T.)—Religious Monomania (J.B.)—Monomania of Persecution—Progressive Systematised Insanity (Paranoia)—Primary Implication—Systematisation—Neuropathic Basis—Secondary Systematised States—Typical Psycho-Neurotic Form—*Folie à deux.*

Amongst states of mental enfeeblement are comprised numerous widely different groups, which constitute the large bulk of our asylum communities, and in which the mental ailment differs in its mode of origin, essential nature, and the characteristic features presented. The term mental "enfeeblement" is perhaps the least objectionable which we may employ for the various groups comprised under this class of mental ailments, but there is a sense in which its application is faulty. The term should, we think, include states of **mental deprivation** only—*i.e.*, states of **acquired defect,** whilst *congenital* and developmental arrest would be more appropriately considered under a distinct category. Idiocy and imbecility would, therefore, be excluded from this class, which would, however, comprise the various forms of monomania, of chronic mania, and dementia.

All instances of mental reduction are, of course, states of mental enfeeblement, and, therefore, in one sense, *all cases* of acute insanity are alike cases of mental enfeeblement, as is the stage of stupor following acute insanity, or an epileptic outburst. We do not, however, extend to this term so wide a significance; we arbitrarily exclude states of *transient* mental deprivation, and limit its connotation to conditions of **persistent enfeeblement,** whether primary or consecutive in their origin. In fact, we comprise under it the incurable terminations of acute insanity—the chronic insane residue which remains, as wrecks remain after the storm ; also, such cases of permanent enfeeblement, as are *not* preceded by acute mental symptoms, *e.g.*, the "primary dementia" of organic disease of the brain—from morbid growth, apoplectic foci, and cerebral *ramollisement*—and the dementia of senile atrophy. Under the respective headings of epileptic, apoplectic or paralytic, senile, and alcoholic insanities, we shall allude to the features presented by the mental decadence accompanying such affections; but we must here devote our attention to a consideration of consecutive dementia as a sequel to the acute forms of mental disease in general.

Number of Cases.	Form of Mental Disease.	PERCENTAGE.			
		Recovered	Died.	Relieved.	Chronic Remainder.
		Per cent.	Per cent.	Per cent.	Per cent.
134	Mania—Simple, ...	61	11·9	11	16
201	Acute, ...	65·5	13·4	8·4	11·9
66	Hysteric, . . .	75·7	3·0	9·0	12·0
46	Chronic, . . .	...	13	36·9	50·0
237	Delusional, . . .	37·0	13·0	18·5	31·0
85	Recurrent, . . .	57·6	8·2	14·0	20·0
46	Puerperal, . . .	71·7	15·2	6·5	6·5
141	Melancholia—Simple, . .	61·7	14·0	13·4	10·6
51	Acute, . .	54·9	23·5	5·8	15·6
299	Delusional, . .	55·5	14·0	14·3	16·0
16	Recurrent, . .	50·0	12·5	12·5	25·0
11	With Stupor, .	63·6	9·0	...	27·2
68	Dementia—Senile, ...	...	60·0	26·4	13·0
28	With Excitement, .	39·0	35·7	10·7	14·2
39	,, Depression, .	41·0	23·0	28·0	7·6
33	Organic, . . .	9·0	54·0	30·0	6·0
121	Epilepsy, ...	11·5	26·4	23·0	38·8
74	General Paralysis, . . .	...	72·9	21·6	5·4
82	Imbecility, ...	...	17·0	51·3	30·7
13	Idiocy, ...	...	38·4	15·3	45·9
18	Chronic Cerebral Atrophy, .	...	77·7	22·0	...
1809					

A glance at the preceding Table, which affords us the results of treatment in the case of 1,809 female patients admitted into the West Riding Asylum, will serve to indicate whence our chronic insane inmates are chiefly derived.

In the Table it will be observed that a large proportion of maniacal and melancholy patients are discharged "relieved," and this class comprises a number of *permanently enfeebled* minds, in which the acute symptoms having subsided, the subjects are safely disposed of under the care and supervision of their friends; hence the chronic remainder in our asylums do not represent by a long way the con-

secutive dementia of acute insanity. Bearing this fact in mind, one may still advantageously compare the total number of chronic cases remaining after maniacal and melancholic seizures respectively—it is then found that out of 815 instances of all the forms of mania, a percentage of 20·6 remain permanently crippled in mind; and that out of a total of 518 instances of melancholia, a percentage of 15·0 remain as a chronic residue.

This is what we might anticipate from our knowledge of the deeper reductions pertaining to the maniacal forms, and confirmatory of it we note a progressively increased tendency to chronic enfeeblement, resulting in the acute, delusional, and recurrent forms, as compared with the simple form of melancholia (*vide* Table). If we summarise results for all forms of mania and melancholia, we obtain the following:—

	Percentage Recovered.	Died.	"Relieved."	Chronic Remainder.
Maniacal forms, .	53·2	11·9	14·0	20·6
Melancholic „ .	57·1	14·8	12·9	15·0

The more unfavourable character of mania depends upon the incurability of its delusional forms; the simple and acute maniacal seizures, if they do not tend to the delusional form, are usually of high recoverability, as indicated by our table; certain forms especially so, as the puerperal and hysterical. Were it not for the large proportion of *such acute cases*, the unfavourable nature of maniacal, as compared with the melancholic forms of insanity, would be strikingly obvious.

Consecutive Dementia.—Ordinary consecutive dementia, however, presents us with a progressively advancing enfeeblement of mind, a complete change in the disposition and character of the patient, a lack of interest in former pursuits and associations, an incapacity for any form of mental effort, a tendency to an automatic routine in the habits of life, and a notable blunting of the emotions. Maniacal or melancholic states occasionally return, and betray, in a marked degree, the incoherence of thought and the enfeeblement of the mental faculties; but, subsequent to such attacks, the mental weakness continues to advance, until it issues in complete fatuity. Yet we find great diversity in the progress of individual cases: in many, the advent of such a mental void, as we have just alluded to, only comes after a very prolonged life, during which they show no mental perturbations, but an apathy and indifferentism, a lack of initiative which renders supervision necessary to provide them with the wants of life; others take a more genial interest in their surroundings, but yet are childish in their actions, are docile and easily led, but subject to great instability if annoyed; in others, again, the brutalising of their nature is more apparent—degraded habits come to the front, vicious tendencies are apparent, but conduct is wholly devoid of all intelligent direction or rational initiative. Many of these chronic dements are utterly lazy, disinclined for any form of exertion, and cannot be induced to employ themselves at the simplest manual labour. They will stand about for hours, slovenly and disorderly in attire, fumbling with their fingers, disarranging or tearing their clothing, and uttering continuously a string of incoherent gibberish. Some of these subjects may have no delusion apparent, as a rule; but yet, at times, a mild maniacal attack may reveal some delirious conception, which again fades away as the excitement abates. The expressionless features betray the lifelessness of mind; or a fixed, hideous grimace, or unmeaning aspects, its unreason.

It would not serve our purpose here to attempt any classification of such numerous and incongruous types as are presented by the cases of chronic enfeeblement amongst the insane; they can only be studied by prolonged clinical observation in the wards of an asylum. Griesinger has, however, distinguished between the class of excitable and that of apathetic dements, and to his vivid delineation of these types we would direct the student's attention.* They represent but **different depths of reduction,** the former being allied to mania—in fact, retaining a certain degree of its mobility as relics of the maniacal condition; the latter being the more profound reduction, in which sluggishness of mind verges upon absolute fatuity. We shall revert to the morbid evolution of these phases of dementia in our section on the morbid histology of the brain.

The transition from acute insanity is by no means always a direct transition to these forms of mental enfeeblement—an intermediate stage of peculiar chronicity often precedes the more profound dementia which we have just considered. To these forms of monomania or delusional insanity proper, we must now revert.

Delusional Insanity.—We have spoken of maniacal states as presenting us with reductions to a stage lower than that attained by melancholic states; and we now come to a group of cases comprising symptoms wholly distinct from those presented to us by the foregoing. This third group lies, so to speak, in the order of dissolutions, on the border-land between the two former. In the first (mania), we noted the general exaltation and the free translation into action; in the second (melancholia), we observed the rise of painful feeling associated with general depression and restricted activity; in the third we find, as often as not, an emotional indifferentism allied with false beliefs of an exalted stamp—a calm, which is, however, ever ready to pass into states of *transient* excitement, on the one hand, or into gloom and despondency on the other. This third group comprises the so-called states of **monomania.** Monomania as a morbid entity must be regarded as a state *evolved out of* melancholic and maniacal perversions—as a special derivative of these conditions, and as one of the **terminations in chronic insanity.** It can be studied to the greatest advantage in association with the preceding forms; nor is it possible correctly to appreciate its significance, if we have not previously analysed the forms of melancholic and maniacal perversions.

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* *Op. cit.*, pp. 340-345.

Genesis of Monomaniacal States.—We have seen that a special feature of maniacal states is the hurry and tumult of the process, and the prevalence of delusive conceptions of a fleeting nature. It is this very rapidity of the cerebral process which accounts for the transient nature of such falsifications; time is required, a certain persistence of impression, or a frequent repetition of the same impression, to form any indelible stamp upon the memory. As stated, one delusion chases another out of the mind in the tumultuous superficial hurry of the maniacal state. The welling-up of feeling, which we have spoken of as the rise in subject-consciousness, finds easy vent in mania in rapid ideation, incessant garrulity, and active movement; yet all maniacs obtain at times full relief in active ideation alone—for the maniacal subject need not be at all times restless, nor need he be garrulous—yet his expression will indicate to us the varying moods and rapid process of incoherent thought going on within. We speak occasionally, but incorrectly, of such cases as instances of *suppressed* mania—there is no mental tension, but complete relief in the active ideational process. Monomaniacal states are essentially those where the rise in subject-consciousness *does not tend to escape in outward action*, but rather to find relief in forms of **perverted ideation;** and herein lies the distinction between the two forms—in monomania there is no longer emotional exaltation and tumult, but perfect calm; the false conceptions arising at these levels of reduction have a far more serious import, since the existing conditions favour their *fixity*. They rise more *definitely* and more *forcibly* into consciousness.

The turbulence of the intellectual life in mania and the heightened mental reflex, we have associated with spasm of the cerebral arterioles, and the resultant quickened circulation in the cerebral cortex; in monomaniacal states, a quiescence of the circulatory current appears coeval with the decline of such exalted cerebral reflex, and we approach the stage of melancholic reductions except for the absence of vasomotor paresis and the stagnant circulation of the latter states; hence, in lieu of a feeling of restricted translation from emotional to intellectual realms, the feeling of freedom and power still predominates. Such freedom, as before stated, finds its output in phases of aberrant ideation.

And yet there are times when the monomaniac realises somewhat painfully a sense of environmental resistance—a sense which must be generated whenever he attempts to put his impossible schemes into practical operation, or tries to convince others of the logicity of his absurd speculations and belief. Especially, however, does this sense of resistance make its appearance in cases of fully-developed monomania, where languor of circulation, induced by cardiac enfeeblement and exhausting affections, such as phthisis, reproduces the melancholic phase afresh. This sense of outward hostility—the irritation and excitement thereby engendered—is a more prominent feature in the *earlier* stage of monomania; and in most cases it is found, in some one or other form, at this period of the disease, as the natural outcome of the antagonism which the subject must recognise as existing between his beliefs and the circumstances around him. It is a feature which indicates the incomplete severance of this affection from the purely maniacal form. As the mania subsides and calm succeeds—as the egoistic feelings predominate more and more, and obtain more complete ascendancy over the intellectual life, the transformation slowly, but elaborately, undergone by the personality is in itself a sufficient answer to all outward antagonism; the all-sufficiency of the new ego, with its wondrous powers, capabilities, and motives for action, dissipates all apparent opposition, or ignores its existence.

It is thus that we find our patients at first, in the early transition-period between mania and monomania, intolerant of contradiction—no opposition offered to their delusive utterances fails to arouse passionate outbursts, violent abuse, and even vindictive conduct; he who risks this often wins for himself the open and long-continued hostility of the patient, at no time a justifiable or politic procedure. At this stage, the deluded subject is loudly assertive of his beliefs, and actively aggressive in his endeavours to carry them into practical operation; in interminable writings, in incessant declaration he will assert his newly-acquired prerogatives; whilst acute hallucinations frequently occur at this period, lending fresh intensity to the drama which he enacts.

In the more confirmed calm of a later stage, a love of **mystic symbolism** is almost invariably apparent; the monomaniac will point to some common-place picture on the wall, expatiating on its secret meaning; he will assume some fantastic badge as the emblem of his exalted dignity—spiritual or temporal; by fantastic gestures or significant movements of the head he will express some meaning hidden from all except himself; or by uncouth scrawls, or geometric devices, he will symbolise Scriptural truths, Biblical records, or scientific discoveries. By such means endlessly diversified, the subjects of monomania beguile their time, and form meanwhile prominent characters in all asylum communities. Their loud threats, their lofty denunciations, their fulminating proclamations, contrast strangely with their impotence in action. They live in an ideal, not a real, world; and are satiated to the full by the mere semblance of authority and power which such expressions conjure up. On this account they are rarely violent or dangerous; they are ruled with the greatest facility, requiring only tact upon the part of the nurse to transform them into most useful and willing helping-hands at various employments. Thus we see the patriarch and delegate of the Deity (*J. O.*) actively at work in the bookbinder's shop of the West Riding Asylum; the Empress of Hermon (*E. P.*) busily plying the needle, trimming the patient's bonnets in the workroom; the "Saviour of mankind" (*J. B.*) taking an active part in the domestic arrangements of her ward; and a notorious admiral who formerly ruled the seas in days gone by, contentedly framing pictures in the joiner's shop.

Cases of Monomania.

In the following case of *J. O.* we see the subject pass through the transition-period from mania to genuine monomania; his case forms a good illustration of the mystic symbolism in which these patients, as we have said above, so frequently indulge:—

J. O., formerly a prison warder in South Wales, has been resident at the West Riding Asylum for nearly nine years. When first admitted he was thirty-six years of age, a well-nourished man of medium height, and free from any bodily ailments. His wife had long recognised his mental failure, but maniacal excitement had now compelled her to place him under restraint. He was at that time, undoubtedly, the subject of fixed delusions: "all human agencies were in league against him, and there was a conspiracy in high quarters to damage him." He had written to the prison commissioners repeatedly about these plots, and was at that time writing a book on "Religion." He talked much about various instruments he had invented, especially "an air and water engine, requiring no boiler," for which he was about to obtain a patent. His condition at this time, and during the following twelve months, was much mixed-up with maniacal excitement—in fact, it was the transition-period to typical monomania. During this period he was often hostile, most unsociable, and utterly indolent; had an arrogant, overbearing demeanour; stalked up and down the wards as though in a position of authority, and grew angry at the most trifling opposition. He was usually reticent, but occasionally talked upon the subject of his inventions and of his experiments upon lightning, which he had conducted "by holding pieces of various metals in his hand during a thunderstorm." He then commenced working on the farm, but would spend most of his spare time reading his Bible—making many differently shaped crosses out of bits of wood, straw, &c., often carrying one in his hand. He declares that he was wrongfully sent here; that he is deputy-governor of a gaol, and possesses the warrant of his appointment; and that he has made numerous discoveries in electricity and magnetism. Since this period he has been regularly employed in the bookbinder's shop, where he is a useful and industrious worker; he is an intelligent workman, and is calm and consistent in his behaviour at all times, outwardly betraying no evidence of the profoundly delusive state under which he labours. He regards himself as a patriarch of the church, and as the appointed of

God to denounce judgments against all evil-doers. Feeling his confinement here inconsistent with these views, he applies to all the crowned heads of Europe for assistance against the persecutions of the medical profession, of whom the writer is the arch-traitor. He writes denunciatory letters to the medical superintendent, calling upon his head the curse of the Almighty, and sends him, every week or so, a pen and ink outline sketch of a coffin, as a last warning, often accompanied by the words, "Behold thy doom;" addressing his missives, "To all whom it may concern," or "Let this find its owner," with some similar suggestive *memento mori*. Occasionally his letters to the medical staff are lengthy and argumentative, freely interspersed with numerous texts, or Scriptural references, containing also words of exhortation and warning, often dictated in the style of the New Testament writings; but it is more usual to find them full of fierce denunciation and threats of divine judgment, as *e.g.*, the following:—

"*May* 6, 1887.

"PROFESSED ENGLISHMEN OR BRITONS,

"I, an English-born subject, J. O., born in the County of Yorkshire, near Huddersfield, Do hereby solemnly declare in the name of 'God,' the Almighty, the Supreme and Invisible Spirit, and pronounce through His Almighty authority, His damnable curses and judgments upon you, and your supposed and so-called Gracious Sovereign and all her subjects, both spiritual and temporal, for this my incarceration in this Asylum or any other.

"J. O., late of Halifax."

In the following case we have a remarkable instance of the transformation into the monomaniacal state upon the occurrence of epileptic seizures:—

J. B. was admitted at the age of forty-two years in an acutely melancholic state. She was of slight build, thin, reduced and anæmic, having been in feeble health since her last confinement, twelve months before. She was not known to be an epileptic. At this period she was greatly distressed by aural hallucinations, and when at home the previous day had heard people moving about beneath the floor of the room she occupied, sharpening knives and saws to murder herself and family; all night long she heard her child crying distressingly on the staircase, and men scraping at the walls of her bedroom. She was firmly convinced that her soul was eternally lost—could see no escape from destruction, and under these impressions she made several desperate attempts at strangulation. She refused food and medicine most persistently, and accused herself of every form of iniquity. This despondency continued for some six months, when the patient had a series of epileptic fits, the character of which was not noted at the time; but now an entire change was inaugurated in her mental life—the depression abated, and she assumed a cheerful aspect; took an intense interest in all around her, and became an active and valued ward-help. The epileptic seizures have occurred ever since, but invariably at night, and with very long intervals, often of years, between the attacks. For many years she has been a typical example of religious monomania. The fits she believes are caused by the "working of the spirit—which has been working very powerfully upon her for some time—because the Father has thought proper that she should bear it for the salvation of the world." She is still in delicate health, and suffers considerably at times from migraine. Always smiling and cheerful, affable with all alike, she is a great favourite in her ward; all who are brought into contact with her are, she believes, made eternally happy through her instrumentality. She is still *in idea* a sufferer—a Christian martyr. "She came here because she thought she had to save all the world. She knows that Christ died to save sinners, but feels that God has given her that power. She thinks that if persons touch her it does them good, and saves them. All who have come here have come through her, and she feels responsible for them. If all their souls rest on her, what an account she will have to give at the day of judgment! She cannot sleep at times because the 'spirit keeps working in her like quicksilver.'"

Thus all former painful mental states have been sublimated into this higher ideal existence. She still answers to her former name, but her personality is, as we see, completely transformed. At times she will state that she feels she is Jesus Christ, that she existed before the foundation of the world, and will cite scriptural passages referring to the Messiah, as applicable to herself. She "loves everybody in the world," and during the evening is often found at the window singing aloud, "Hold the fort, for I am coming," in shrill accents, with the object, as she says, of "helping those outside." She has a gentle, quiet, inobtrusive manner, has the sweetest disposition, spends much of her time perusing her Bible, and is often found seated musing, with her hands crossed upon her breast, and an expression of peaceful resignation stirred into beaming animation when she is addressed.

E. T., aged forty-six years. This patient, who is a married woman, the mother of six children (the youngest born four years prior to her admission into the asylum), was then suffering from her first attack of insanity of a few weeks' duration only. She had been confined to bed for twelve months, suffering from bronchitis and emphysema, and troubles incidental to the climacteric period.

The history of her case was one of depression, groundless fears, and delusions of suspicion fostered against her family, who she believed conspired to poison her; under the influence of these fears she obstinately refused food, and passed restless nights, sitting up in bed continually praying.

She was regarded as at the climacteric. Family history devoid of neurotic taint. On admission she was extremely thin and wasted. She is short of stature, bony, and of a somewhat masculine type; she has light blue eyes, a sharp penetrating glance, and a suspicious demeanour. States that her husband, daughter, and neighbours have conspired to remove her from her home, that her daughter has the power of witchcraft, and can appear in various forms; that her family and neighbours introduce saliva and other disgusting matter into her food; and that she has been given gold dust and serpents to swallow. She stoutly maintains these statements, and declares that on the previous night she believed herself to be in labour of serpents. She hears her son and daughter whispering through the wall, and addressing her by foul and abusive names. No visual hallucinations are at present obvious. The physical examination revealed general bronchitis with emphysema, but no consolidation or evidence of incipient phthisis, such as her appearance suggested.

Steady improvement occurred in her case, her delusions faded away, and within a month she was regarded as convalescent. Then occurred a sudden relapse, in which maniacal excitement replaced the former mental depression, and a downward career of mental reductions has ensued unchecked ever since. Her general health underwent marked improvement, but she always remained pale and anæmic. Her excitement was characterised by loud, abusive, and blasphemous language to all around her, and by a hostile demeanour and threats of violence to those who approached. Little or no abatement of her excitement took place under treatment by succus conii, bromide, with Indian hemp, opium, or hyoscyamine. Her condition, during the five years succeeding the onset of attack, was that of typical monomania. She would sit isolated from other patients in a recess before a window, choosing a position where a portrait of one of the Royal Princes hung opposite her. Here, decorated in fantastic attire, her hair adorned with feathers, coloured ribbons, or mock diadem, and her dress decorated with coloured devices, all of which had some mystic symbolism to herself, she would sit in state, the embodiment of pride and arrogance. From hence she issued her mandates to the world around, or met those who approached her with scornful defiant gaze, together with a torrent of lofty abuse and imperative orders to withdraw from her presence. Occasionally she would deign to expatiate on her lofty rank, would point to the portrait of the young Prince, and speak of herself as the Empress—his mother. Her conversation was now very incoherent at times, but was invariably tinctured by her grandiose delusions. She frequently complained of sudden sharp pain in the side, which she attributed to having been shot there by the medical officer. On one occasion she was heard to utter a loud piercing shriek, and was observed, transfixed with horror, gazing at, and pointing to, an imagined tragedy, which was being vividly enacted before her—"See! see!" she cried, "they have the knife in him—look at the blood;" then she fell back in her chair and laughed with derisive laughter.

Ten years after her admission her habits are noted as similar, and the mental features as unaltered.

Twelve months later, evidence of phthisis was revealed, and a slight attack of hæmoptysis occurred. She complained of pain in the lower dorsal region, and asserted that "that part of my spine has been cut out, and made into jelly; all parts of my body have been made into jelly and thrown on the floor; I've been a doctor 300 years."

Latterly, she has broken down completely in health; phthisical symptoms have been for some time prominent, and occasional hæmoptysis has occurred. Completely bed-ridden, and a great sufferer from exacerbations of her chest symptoms, much prostrated in health, pallid and emaciated, she still asserts her royal prerogative, and insists upon being addressed by her formal title; she issues her mandates to her courtiers, princes, and statesmen, with gestures of mock authority; and still, at times, becomes irate at the least appearance of opposition. There is now considerable mental enfeeblement; increasing incoherence of ideas, and a tendency to substitute unmeaning words, and interpolate them in her sentences so that they constitute at times a confused and unintelligible jargon. She is now tractable and devoid of all the repellant features characterising the early stage of her alienation—amiable, as a rule, but still subject to mild outbursts of irritability and excitement, in which her delusional notions become very prominent.

Both these cases, we observe, are of many years' standing; in fact, monomania is a most chronic form of insanity, gradual in its inception, and very slowly progressing towards general mental enfeeblement; the coherence of former associations becomes successively loosened, whilst the fictitious personality persists and erects itself skeleton-like amidst the ruins of mind.

Progressive Systematised Insanity (Paranoia).—The clinical features which first appear to have suggested the constitution of a distinct morbid entity to which the terms "Verrücktheit," "primare

Verrücktheit," "Progressive Systematised Insanity" were applied were:—

(*a*) A Primary Implication on
(*b*) A Neuropathic Basis;
(*c*) A Systematised Delusional Perversion with
(*d*) Progressive Mental Enfeeblement and Incurability.

What value ought to be assigned to each of these terms in the constitution of a nosological entity? Let us endeavour to weigh their significance for purposes of classification.

Primary Implication.—By this term is implied a specialised insanity or one *not preceded* by a generalised insanity, such as mania or melancholia; in other words, a primary alienation not based upon an affective disorder; an alienation gradually evolved, in which the earliest symptoms may be most obscure and indefinite, but eventually culminating in complete intellectual downfall. With regard to the value of this feature, it may be stated that, whilst all forms of insanity appear to be preceded by more or less affective disorder, the intensity of such emotional disturbance varies with each case in every possible degree; so that it is difficult, nay impossible, in many cases to state whether the insanity was truly primary in this sense, or secondary to an affective disturbance. Taking also into consideration the doubt so often introduced by want of observation, or by lack of intelligence on the part of the friends, we are still more inclined to assume that a somewhat arbitrary value has been assigned to this feature for purposes of classification.

Professors Wille and Meynert both affirm that there are not many cases in which it is difficult to determine, in the early stage, whether they belong to primare Verrücktheit, or to melancholia (*Hack Tuke*).

Since the days of Griesinger, who emphatically declared that emotional disorder *always preceded* systematised insanity—that the latter was always secondary—German alienists have shown a complete change of front, and now subscribe largely to the opinion long held by French authors as to the existence of a *Primary Verrücktheit.*

From what has already been stated by us as to the all-prevalent feature at the onset of mental disease (p. 153) it must be inferred that a disorder of feeling precedes and accompanies all forms of insanity; and that a primary implication of the intellect, strictly so to speak, cannot be logically assumed to exist. In all such cases as are quoted as types of primary systematised insanity we note the affective disorder in early stages and throughout the disease; the melancholic gloom, or other disorder of feeling, from the earliest days may be mild and, as it were, drawn-out fine, yet this inobtrusive character does not warrant us in asserting that the morbid syndrome has not been evolved out of the moral nature.

Systematised Delusional Perversion.—Here again the value of a systematised process for differential groupings appears to be open to question, since systematisation is a feature prevalent in almost all progressive forms of mental alienation—*i.e.*, where emotional storms are in abeyance. It is essential to the establishment of a systematised insanity—to the evolution of, *e.g.*, that extraordinary morbid development we know as monomania—that the morbid process should work to its destined end in an atmosphere of comparative calm; in fact, all progressive insanities, if not accompanied by much tumult or acute disturbance, tend towards systematisation. At all periods of life, in early youth, during adolescence, middle life, and even in senility we may frequently trace this tendency to systematisation—much seems to depend upon the absence of emotional turmoil. This insistence upon the systematised nature of the insanity appears to us, therefore, as a simple statement over again of the well-recognised fact, that in all morbid reductions of the mental life the interpenetration of the morbid factor is favoured by emotional calm; that whenever a morbid process involves the intellectual element of mind, it is clear, defined, and more systematised—the less the perturbation of feeling and emotion. This is but a primary fact in psycho-physics, and a part of the natural history of insanity in its *ensemble*, but scarcely seems to warrant the position assigned it as the essential feature of a specific entity. When the psychologist tells us that the more there is of feeling the less there is of cognition, and *vice versâ*, he expresses the law which is the basis of all systematised psychoses; the greater the emotional turmoil, the fewer and more unsubstantial are the possible coherences established in the intellectual sphere, and, therefore, the more dissoluble and transient the delusive concepts. Systematisation in mental disease has, therefore, for us a far more general value, and cannot thus forcibly be detached as the prevailing feature of any one artificially constituted syndrome.

The Neuropathic Basis.—Here again appeal is made to a very general accompaniment of insanity, not necessarily of the so-called systematised groups. Its value here seems to us chiefly to depend on the explanation it affords of the pathogenesis of the so-called Primary Insanities, as offering an explanation of the gradual onset without acute or generalised symptoms. Our views on this point have been given upon a former occasion, and we shall here merely quote the opinions then arrived at:—We held that there was no form of mental derangement which had not some degree of affective disorder as its accompaniment; that a certain section of the degenerative class had arrived at a level—through *former parental emotional storms*—at which any notable affective disorder was in most instances no longer possible; that what was termed "Paranoia" was simply delusional insanity engrafted on a degenerative or psychopathic basis; that there were reasons for believing that systematisation depends mainly upon the incidence of insanity upon a psychopathic subject, and that too much emphasis has been bestowed upon it as a differential feature in the various forms of insanity.*

Referring to cases of criminal paranoics we have elsewhere stated that it "is open to suggestion that such subjects are at the least the relics of a neurotic ancestry in which the *more acute storms* of disease, to wit—acute alcoholism, convulsive epilepsy, impulsive forms of insanity, &c., have already passed over the parental stock, whilst the *later stages* of such nervous affections betray themselves in the mental enfeeblement of their progeny.† In this particular it is gratifying to find our views in complete accord with those of Italian alienists.

If there be any excuse for the use of the term "paranoia" as a synonym for "Verrücktheit" and as connoting a group of primary systematised insanities, there certainly can be none for so-called "*Secondary Paranoia*," which would embrace all systematised delusional insanities evolved out of a generalised insanity—and which does violence to the prime characteristics of the disease known as paranoia—viz., the *primary* mode of onset of the psychosis. These forms of delusional insanity have long been studied by English authorities; they have been spoken of as rare by Mendel, and, according to Regis, have scarcely been studied at all in France. ‡

Just as unmeaning and illogical then becomes the term "*Acute* Paranoia," by which is implied a transient systematised insanity, of a curable nature, and the existence of which has been denied by Tanzi and Riva, Krafft-Ebing, and others. French authors have wisely abstained from recognising its existence, since the usual implication of the term "paranoia" would negative its possible occurrence. It appears to us, therefore, that we might with far greater consistency limit the term "paranoia" (if it is indeed necessary to retain it) to what has been described by the Italian School as **degenerative paranoia** and the **primary psycho-neurotic form,** eliminating altogether the so-called acute and secondary paranoias.

The term "paranoia" is ill-chosen and, etymologically, has not the significance of that far better term which it tends to displace—"primare Verrücktheit," or primary systematised insanity; these latter at least have the merit of *defining* the disease implied, whilst the former has led to still greater confusion than previously existed, and appears to have been introduced to avoid the awkward implication of *primary* attached indelibly to the term "Verrücktheit"; it has led also to the subdivision into numerous groups of questionable value of the classical "delusional insanity" of English authorities.

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* *Brit. Med. Journ.* (Meeting of Psychological Sec.), August 20th, 1892.

† "The Origins of Crime" (*Fortnightly Review*, September, 1893).

‡ *Mental Disease* by Regis, translation by Bannister, p. 166.

Our own view is in favour of substituting for these terms—

(*a*) Primary systematised insanity of the degenerate.

(*b*) Typical primary systematised insanity (*i.e.*, psycho-neurotic);

yet always with the proviso that the term "primary" may not be held as strictly correct or even justifiable, implying merely a proportionately mild emotional reaction in the one case, and, in the former case, that the original acute psychosis is still to be traced in the ancestry—a view also accepted by the Italian school of psychiatry.

We shall describe in detail here only the typical primary systematised insanity:—

Typical Systematised Insanity (Psycho-neurotic).—The evolution of a typical case of progressive systematised insanity presents three stages of varying duration—(*a*) Hypochondriasis with suspicion; (*b*) Persecutory insanity; (*c*) Transformation of the personal identity.

(*a*) The subject in this early stage betrays a notable hypochondriasis, a self-analytic tendency, in which the victim refers all his troubles to some outside agency. All his feelings—bodily and mental—are subjected to a rigid self-analytic process; trivialities are exaggerated to undue dimensions; bodily sensations are criticised with a morbid intensity; his ideas and mental faculties all appear to have undergone some peculiar and mysterious change; his environment looks altered, and he reads in the gestures, looks, signs, and words of those around him a special reference to himself. With this weakening of object-consciousness and intensified egoism, suspicion of the environment grows apace, depression and gloom supervene, reticence and brooding indicate the gathering distrust and hostility to the outer world.

(*b*) Hallucinations, almost invariably aural, now appear upon the scene; smell, taste, tactile and general sensation may all be involved; but visual hallucinations are remarkable by their absence. The hallucinations of hearing may be unilateral, bilateral, or antagonistic—the "epigastric voice" may be heard, or his thoughts be repeated audibly within his brain, and any of the varied forms of hallucinations, including the sexual already described, may prevail; in all cases they suggest the malign and hostile character of his environment. Not only does he find his thoughts echoed within him, but also his most secret feelings are known to and reflected by those around him. Some mysterious power seems to have gained access to his mind, regulates his mental life, enslaves his will and speech, so that he is driven to think and utter ideas not his own. Electricity, galvanism, hypnotism, telephonic, telepathic, and other agencies are evoked to explain these mysterious powers exerted over him by his enemies; and elaborate sketches are often drafted which detail to his satisfaction the methods whereby he is subjected to such torture. The numerous phases presented by the several forms of monomaniacal perversion all betray the same progressive systematisation of the delusive concepts; the prevailing hostility of the environment and resultant distrust; and, finally, lead to the climax in the third stage, viz.:—

(c) A transformation of the personality which is introduced by mystic and symbolic references, by the creation of unmeaning words (neologisms) to express what they otherwise cannot convey; and the final apotheosis of the mind into the vagaries of ambitious insanity.

We have here described what is regarded as a typical case of so-called "paranoia" or primary systematised insanity: it will be apparent to all who have read the preceding chapters on Mental Depression, Exaltation, and Enfeeblement that the features now presented are those which characterise all forms of persistent delusional insanity or monomaniacal perversion. Degenerative paranoia is, therefore, nothing more than delusional insanity arising in a degenerate subject, and consequently stamped with peculiarities which more or less prevail in all the psychoses of the degenerate. Psycho-neurotic paranoia is the typical course pursued by all delusional insanities; whether as the outcome of a generalised insanity, or whether it be not, is a mere accident, and apparently of little moment as regards the ultimate course and issue of the case. A systematised insanity absolutely identical in all its stages with that just described is by no means of rare occurrence as the outcome of adolescent mania; similarly also of alcoholic insanity; both would be held to be genuine cases of secondary systematised insanity—not a partial but a generalised insanity.

Folie à deux.—Systematised delusional insanity is the form which, far more frequently than any other, appears to give rise to so-called "communicated insanity" or **Folie à deux**; a condition where intimate association between an insane and a sane individual leads to a *similar* mental disturbance in the latter. The absence of acute, generalised mental disturbance, the calm reasoning insanity, the systematised character of the delusive beliefs, and their steady and gradual evolution, all tend insidiously to produce their morbid effects upon the mind of a strongly neurotic subject, closely associated either by ties of relationship or as companion with the alien. The malady thus aroused is usually identical in nature, and exactly similar delusive concepts become manifest unless such association be discontinued. The offspring of a highly neurotic stock may thus show in adult life the arousal, first in the one, then in the second, third or more, of a similar systematised insanity; or a deluded husband may arouse the same form of disorder in a neurotic wife, or *vice versâ*. Where several members of the same family are affected, and the malady is similar in character, we must by no means conclude that they are necessarily instances of *Folie à deux*; we must carefully distinguish betwixt direct transmission from a neurotic stock, and the morbid effects of association betwixt individual members of the family. In fact, it is only when it has been clearly shown that there is a direct morbid influence of the one upon the other, that intimate association has existed, and that evolution of symptoms pursues the same course in both individuals, we can reasonably assume a psychic infection. When individuals *not related by consanguinity*, but otherwise closely associated, betray a *similar delusional insanity*, aroused upon a psychopathic basis, we may regard the case as strongly suggestive of *Folie à deux*; yet, no hasty conclusion should be arrived at, even here, since much may be attributable to identical conditions of life, corresponding lines of feeling and sentiment, and, especially, similar morbid or vicious habits, in neurotic subjects.

That the fear of such developments is but a remote one, even in the opinion of those who fully recognise the existence of this malady, is sufficiently evident in the promiscuous association of the subjects of systematised insanity with all classes of the community in our large asylums. Whatever plea has been advanced for segregation of different classes of the insane, certainly that which has received the least emphasis has been the plea for the avoidance of communicated insanity through too promiscuous association.

RECURRENT INSANITY.

Contents.—Definition—Establishment of Labile Equilibrium—Prevalence at Sexual Decadence—Heredity—Influence of Neurotic Heritage and of Ancestral Intemperance—Atavism—Recurrence in the Congenitally Defective Subject—Morbid Excitement and the Moral Imbecile—Alternations of Excitement and Stupor—Hysteria and Menstrual Irregularity—Eroticism (*A. S.*)—Recurrence in Adolescence (*M. C. W.*)—Recurrence at the Climacteric (*H. O.*)—at the Senile Epoch (*J. S.*)—in Puerperal Subjects (*M. B.*)—in Traumatic Insanity (*B. L.*)—Morbid Impulsiveness—Hallucination and Delusion (*J. B.*)—Circular Insanity—Prognosis—Treatment.

All forms of insanity are prone to recur; from whatever source the unstable condition of the nervous centres is derived, whether from inherited neurotic tendencies, acquired vices, or physical ailments, all alike (even the most recoverable forms) have such a predisposition, intensified by the occurrence of an attack. It is a general law that the more frequently a centre discharges its energy, the more sensitive to excitation becomes the mechanism, and the more readily the discharge repeats itself. Hence, the extensive cortical discharges which account for the reductions of insanity will, even in the most complete recoveries, tend to foster a similar hyper-sensitiveness, and a **labile equilibrium** of the parts **previously involved.**

By **recurrent insanity** we mean a type of mental disturbance in which there is an establishment of this **labile equilibrium**; and the conditions under which such recurrence is brought about, together with the essential nature of the attack, form the subject of our inquiry. In the first place, it must be remembered that a neurotic inheritance, however strong, does not *necessarily* result in recurrent insanity; and, in the next place, it should be noted that simple relapses of insanity, which may occur at different periods throughout life, do not imply the existence of the neurotic type here alluded to as recurrent insanity.

Recurrence, with long intervals of repose, is not the characteristic of this type, but rather the rapid succession of attacks, each followed by an apparent complete convalescence. "Nothwithstanding the authentic instances of recurrent insanity showing intervals of lucidity for very long periods, so that the disease is known to be dormant for years, it is by no means to be inferred that every case that is a second attack belongs to such a category" (*Sankey*).* A large section of the insane community is, therefore, constituted by these unfortunate ones, who pass many years of their life between an asylum and their home during frequent alternations of sanity and insanity. Those not conversant with statistics of insanity have but a faint notion of the miserable existence of such victims. The following scheme of some fifty recurrent cases amongst women will exhibit this fact in a striking manner.

When dealing with the insanity incident to the periods of puberty and adolescence, it will be seen that recurrences are not frequent in the proper acceptation of the term; up to the stage of complete convalescence relapses are peculiarly prone to occur, but, once the cure is complete, a recurrence of insanity is not frequent, sixteen instances only of a third or fourth attack being given in 277 cases.

Recurrent forms of insanity are far more prevalent in adult life, and increase gradually towards the decline of manhood and womanhood. In men, quite one half the cases of recurrent insanity occur after forty years of age; and out of a total of 66 individuals so affected, 49 had passed their thirtieth year of life; similarly in women, we find that nearly half the cases cover the period of life between forty and fifty-five, which may be safely taken as the limit of the climacteric period. In fact, the period of life between forty and sixty years in the female is peculiarly susceptible to this form of mental derangement, being the period involved in sexual decadence and the advance of senility. In man this feature is not so apparent, there being other influences, as we shall see later on, which tend to beget in him such recurrent attacks at a somewhat earlier period of life.

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* *Lectures on Mental Disease*, p. 179.

TABLE OF FEMALE RECURRENTS.

Number of Attacks.	Occurring between the Age of	Representing Interval of	Number of Attacks.	Occurring between the Age of	Representing Interval of
5	16 and 32 yrs.	16 yrs.	Several	31 and 36 yrs.	5 yrs.
3	17 ,, 20 ,,	3 ,,	5	32 ,, 48 ,,	16 ,,
3	17 ,, 21 ,,	4 ,,	8	32 ,, 42 ,,	10 ,,
Several	18 ,, 30 ,,	12 ,,	5	33 ,, 37 ,,	4 ,,
3	19 ,, 24 ,,	5 ,,	4	35 ,, 50 ,,	15 ,,
3	20 ,, 21 ,,	1 ,,	6	35 ,, 53 ,,	18 ,,
3	20 ,, 25 ,,	5 ,,	4	35 ,, 48 ,,	13 ,,
3	20 ,, 33 ,,	13 ,,	3	39 ,, 46 ,,	7 ,,
3	19 ,, 28 ,,	9 ,,	5	38 ,, 50 ,,	12 ,,
4	24 ,, 44 ,,	20 ,,	3	38 ,, 40 ,,	2 ,,
3	23 ,, 25 ,,	2 ,,	4	38 ,, 56 ,,	18 ,,
5	24 ,, 36 ,,	12 ,,	3	40 ,, 45 ,,	5 ,,
4	25 ,, 35 ,,	10 ,,	3	42 ,, 59 ,,	17 ,,
3	26 ,, 40 ,,	14 ,,	7	43 ,, 53 ,,	10 ,,
5	28 ,, 45 ,,	17 ,,	5	43 ,, 58 ,,	15 ,,
4	29 ,, 42 ,,	13 ,,	Several	43 ,, 58 ,,	15 ,,
5	29 ,, 52 ,,	23 ,,	6	42 ,, 51 ,,	9 ,,
3	29 ,, 33 ,,	4 ,,	4	44 ,, 58 ,,	14 ,,
3	30 ,, 41 ,,	11 ,,	3	47 ,, 55 ,,	8 ,,
4	30 ,, 44 ,,	14 ,,	3	46 ,, 50 ,,	4 ,,
4	30 ,, 42 ,,	12 ,,	4	48 ,, 63 ,,	15 ,,
6	30 ,, 43 ,,	13 ,,	5	50 ,, 53 ,,	3 ,,
3	30 ,, 35 ,,	5 ,,	3	50 ,, 58 ,,	8 ,,
3	30 ,, 42 ,,	12 ,,	5	51 ,, 54 ,,	3 ,,
3	30 ,, 35 ,,	5 ,,	3	54 ,, 56 ,,	2 ,,
4	30 ,, 55 ,,	25 ,,	Total, 50 Persons. 193 (+Several) Attacks.		

RESPECTIVE AGE IN QUINQUENNIAL PERIODS, IN 164 CASES OF RECURRENT INSANITY.

Age	Males.	Females.
Up to 25 years.	10	9
,, 30 ,,	7	8
,, 35 ,,	7	9
,, 40 ,,	9	8
,, 45 ,,	7	21
,, 50 ,,	9	9
,, 55 ,,	5	13
,, 60 ,,	4	17
,, 65 ,,	3	4
,, 70 ,,	5	...
	66	98

Who are the subjects most liable to this form of mental disturbance? They have a strongly stamped **hereditary history of insanity;** the parentage, when facts are procurable, revealing attacks of insanity often along both paternal and maternal lines. It is also notable, that in a large proportion of cases, we find the history of ancestral insanity attached to the grandparents, or the collateral line of uncles and aunts, significant of a more remote origin for the neurosis. The actual proportion of cases revealing strongly marked hereditary features (often involving several members of the subject's ancestry) amounts to 36 per cent.; but, in 12·5 per cent. only was it discoverable that the subject's parents had been insane.

In the next place we observe that other neuroses, notably **epilepsy,** are absent in the antecedent history. Chorea, hysteria, epilepsy, hemiplegic seizures are prone to occur in the ancestry of a certain class of the insane, as was seen to be the case in the insanity of female adolescents, where 20 per cent. revealed this predisposition; but such a neurotic history is attached to only 4·4 per cent. of the recurrent forms of alienation.

Again, **parental intemperance**—a potent source of all forms of convulsive neuroses—is revealed in 11·1 per cent. (males 8·9, and females 12·6), or *over two-thirds* the proportion of cases shown by adolescent forms of insanity; and in 80 per cent. of such instances of parental intemperance, the *father* was at fault. This fact is a suggestive one, and the question naturally arises—why one form of insanity should appear, as the result of an insane inheritance, and another as the heritage from epileptic parents or grandparents, or as the outcome of parental drink? If we accept, as we have reason for so doing, the dictum that the hereditariness of insanity, like the heredity of other pathological tendencies, is restricted by sex and age, it may reasonably be assumed that the neuroses of early life—chorea, hysteria, epilepsy—will be especially prone to re-assert themselves also at a *similar epoch* in the life of the offspring; and that, therefore, an epileptic father or grandfather who *became epileptic* at *puberty* will be liable to transmit to his sons a morbid tendency which appears as epilepsy or the like at the adolescent period. Insanity, on the other hand, is not a disease of *early* years, and, as we have seen, is far more frequent towards the middle period of life; hence we might expect its appearances as an inherited affection to be regulated by the same laws. This is seen to be the case with the recurrent form, which is strongly inherited, and which conforms to the law of insanity in general, in being most prevalent at the middle epoch of life. Adolescent forms, however, must be differently accounted for, and may indeed with justice be conceived of as the morbid expression of an inherited neurosis of the epileptic type—epilepsy in the collateral or direct line tending to issue in insanity; often even by atavic descent. It is generally conceded that alcoholic craving is often an *inherited condition*, as in the form of "dipsomania;" and that parental intemperance frequently results in the imbecility, idiocy, epilepsy, or deaf mutism of the offspring—all, we observe, indications of arrested development or disease in *early* life. To this category we may add adolescent insanity, which is especially apt to be engendered in the offspring of those addicted to heavy drinking, under certain physiological conditions and the operation of other excitants.

To revert, however, to the recurrent form—the heredity observed in such subjects is more often **atavic** than direct—its frequent appearance in the collateral line of uncles and aunts, being strong presumptive evidence in favour of an atavism even when no other record exists. Its comparatively later development than the adolescent form appears to be governed by the law of limitation by age, which is enforced in most hereditary affections; the ancestral affection occurring in adult life tends to reproduce itself at the same epoch. Parental intemperance declares itself almost exclusively on the father's side, but is by no means a prominent predisposing element. The neurotic temperament of these subjects is revealed in an undue excitability, and a defective moral control, exhibited often in ungoverned passion, and generally mobile emotional states; occasionally, such lack of control amounts to mild forms of imbecility of the moral type—congenital defects occurring in some 12 per cent. of such cases. For the first start in life, such organisations may readily adapt themselves, and the period of puberty and adolescence passes by without serious risk; but, as the complexity of life increases in the ever more complex environment, corresponding developments do not occur, and adaptation is at fault. The organism but awaits some exciting cause which, as with a fulminate, determines the attack of insanity. If a female, the period of gestation or parturition may so act, or, still more forcibly, the epoch of the menopause; if a male, alcoholic indulgence is a most potent agency in causing the further reduction which issues in acute insanity.

The climacteric, as we have already stated, is a period prone to induce and foster a craving for stimulants; and hence, we find that 13·7 per cent. of female recurrents were addicted to intemperate habits, whilst 30 per cent. of the male recurrents had succumbed to this vice. In short, the subjects prone to recurrent insanity are, in general, congenitally predisposed by defective mental organisation, and inherit a strong parental or atavic tendency to insanity, which usually appears upon the indulgence in alcoholic stimulants, or at the later critical epochs of life—notably the climacteric and senile decrepitude.

Dr. Sankey, on the other hand, regards the periodicity of recurrent insanity as bringing this disease into close alliance with epilepsy; his statement is to this effect:—"By the very character of periodicity (a character of the utmost importance in their pathology), they are allied to epilepsy, and in certain cases actually terminate in well-marked symptoms of that disease; especially when our views of epilepsy include all the phenomena and variation of the *petit mal*, now generally classed with true epilepsy." *

Nature of the Attack.—The seizure varies as to its symptoms and course with the exciting cause at work, and the period of life when it occurs. There may be mild maniacal excitement, without obvious delusional perversion; or the attack may be characterised by delusions of suspicion and persecution; or by an ordinary *lypemania*, with delusions of a depressant nature. Maniacal conditions certainly prevail in the earlier and later periods of life—in adolescence and in senility; whilst the climacteric cycle usually calls forth emotional depression and melancholic delusions. Of subjects prone to *recurrent* seizures of mania or melancholia, the more important are cases of

- Congenital mental defect.
- Adolescent insanity,
- Alcoholic insanity (acute).
- Traumatism (cranial injury).
- Insanity with menstrual derangement.
- Puerperal insanity.
- Climacteric insanity.
- Senile insanity.

Epileptic subjects, in whom recurrent seizures are frequent, are necessarily excluded here by their intrinsic importance, and will be considered apart.

Recurrence in Congenitally Defective States.—Those whose mental organisations are congenitally defective in both sexes are proverbially subject to passionate explosiveness, to rapid alternations of mood, and to other indications of great instability. Such cases are often misjudged, the normal undisturbed state being one of striking placidity and great amiability, which seems to render it highly improbable that the passions will so readily assume the opposite extreme; but, so it is, that such extremely amiable natures will pass, upon the most trivial disturbance, to a bitterness and a passionate demonstrativeness often exhibiting an inherent cruelty and viciousness.

With lack of inhibitory "staying" power, such individuals, as before stated, meet a severe trial during the adolescent period of life; but, if they do not succumb to insanity at this epoch, they are still subject, upon the occurrence of trivial agencies, to an attack of insanity at any subsequent period of life.

The agencies which are thus potent towards such an issue are alcoholic and sexual excess, masturbation, and indulgence in morbid excitement of any class. Masturbation, especially, lays the groundwork for an attack of insanity by the nutritive changes induced in the nervous centres—their exhaustion and the ultimate impoverishment of blood. If this vice be associated with alcoholic indulgence, the effect is vastly augmented, and the worst forms of recurrent insanity occur. Another frequent source of the attack is the powerful influence of morbid emotional excitation—sensational plays, sensational literature, "revival" services, "salvationist" crusades; all have much to answer for in their effect upon the **moral imbecile,** and those lacking in moral control. Menstrual derangements, again, foster in the congenitally defective an explosiveness which may issue in an attack of insanity. In all these cases the agency, whether it be menstrual irregularity, masturbation, sexual excess, alcoholic intoxication, or undue nervous excitement from moral causes, acts by occasioning a malnutrition of the central nervous system, already predisposed to insanity through a neurotic inheritance, expressed in a defective mental organisation.

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* *Loc. cit.*, p. 179.

In the male subject, so constituted, the attack of insanity is almost invariably one of excitement, characterised by noisy, boisterous humour, mischievous conduct, destructiveness, viciousness, and outbursts of violence. Diurnal quiet often alternates with nocturnal excitement—the nights, in such cases, being spent in noisy, incoherent rambling, and often prolonged insomnia. Frequent relapses of excitement are prone to occur before convalescence is more permanently established. Should masturbation complicate the case, the subject becomes a prey to delusions of suspicion, and **alternations of excitement and stupor** will often take place.

In the female subject, the attack of insanity occurring is also one of acute excitement, where mild forms of moral imbecility or naturally defective inhibition are maintained. The type is usually that of so-called **hysterical insanity,** reproducing, as it does, many prominent hysteric symptoms.

The typically hysteric subject is generally the subject of **menstrual irregularities,** as in the following case:—

A. S., aged twenty years, mill-hand, admitted May, 1870. A cousin was insane and epileptic. This patient was, in 1868, placed in Morningside Asylum, and remained there five months, being discharged at the request of her friends, although probably not recovered. Her second attack, for which she was treated here, was characterised by violent eroticism; she conversed incessantly on marriage, &c. Its duration was short, but she was not considered sufficiently stable to be discharged under nine months. The menstrual function had been regularly performed as regards time, but in amount variable, occasionally rather profuse, more often there was amenorrhœa. In less than two years she again required restraint; the catamenia having ceased, she, about a month afterwards, became restless, sleepless, and excitable, prone to destructiveness, and very indecent both in speech and demeanour. When admitted, she was somewhat maniacal, but intensely erotic—betraying much perverted sexuality. Showed evidence of the existence of aural hallucinations. She improved, and was discharged under a twelvemonth. A fourth occurrence of insanity took place twenty months later, the catamenia being on this occasion regular. Only slight premonition was given, and the patient became suddenly excited, violent, incoherent, with much religious matter mixed up in her ravings; but the sexual feeling only displayed itself once in three weeks, during which time the mania subsided. Eroticism was alone manifested in connection with irregularity of menstrual function, which occurred later; it did not cease until the catamenia had resumed their normal characters. Discharged in thirteen months.

Another relapse, eight months subsequently, was purely maniacal in kind; there was rambling at first upon religious topics, slight or no evidence of eroticism, and no added irregularity of menstrual performance—which, it was stated, generally erred on the side of insufficiency. Seven months accomplished a cure. The sixth and last admission occurred eight months later; menstrual derangement had again preceded. The condition was one of simple mania, with great religiosity, but without sexual characters, predominating. Convalescence occurred immediately the catamenial irregularity was remedied. Sent out in seven months.

Recurrence in Adolescent Cases.—Adolescence occasionally ushers in recurrent attacks of mania—three or four such seizures, between the ages of seventeen and twenty-five years, being sometimes witnessed. In all such instances, the symptoms reproduce over and over again the features (already delineated) of insanity occurring at this epoch. Recurrent mania originating during adolescence is of very ominous portent; the prognosis is exceptionally unfavourable, in so far that it indicates, for a large proportion of cases, a congenitally defective mental organisation; that many others are doomed to successive attacks beyond this period of life; and that the rest remain chronic residents of our asylums, or are discharged as partial "recoveries" only—or their recovery, if at all complete, takes place after a protracted illness, often embracing successive relapses.

M. C. W., aged eighteen, single; a tall, well-proportioned girl, of somewhat delicate aspect, feeble muscular development, dark brown hair, light blue eyes, complexion fair, expression bright, animated, and intelligent. For some time past she has been in delicate health, and is distinctly anæmic. She brings with her a strong neurotic heritage; her maternal grandmother was twice under treatment at an asylum, her mental balance overthrown each time by "some love affair;" her own mother is highly eccentric; and she herself has been regarded as very unstable, flighty, and erratic. The catamenia have been excessive of late, and of fortnightly occurrence. For a month prior to her attack, the patient had been attending exciting religious services, "revival meetings," and had been excited over these subjects, talking much in a religious strain. The attack occurred a few days before admission; she became acutely maniacal, garrulous, and incoherent. On removal to the workhouse infirmary, she tore down the pads of the padded room, was extremely violent, and her conduct most outrageous. When brought to the asylum, the maniacal condition was still acute; she was good-tempered, jovial, mischievous, talked incessantly, and gambolled from subject to subject, but could carry on a connected discourse on closely questioning her, and insisting upon a reply. She had a flippant air, was pert in her remarks, and shrewd. Appeared quite unaware of the nature of her surroundings. Left to herself, she talked incoherently, interspersing her remarks with frequent allusions to "angels, hell, and devils," saying that she heard "trains whistling telegrams to heaven, when at the workhouse."

Half an ounce of the succus conii was ordered twice daily, and chloral was given occasionally at night to secure sleep. In four days it is noted—"Much more composed and rational; sleeps well, and is trying to employ herself usefully; has not yet lost the flightiness of behaviour and demeanour; appetite good." In a fortnight she was in the "convalescent" ward. A month subsequent to her admission *a complete relapse occurred*, characterised as before by noisy, boisterous behaviour, great hilarity, pertness, and occasional insolence; her nights were not disturbed by excitement. Conium (succus conii) was given in one ounce dose twice daily, and towards the middle of the month the excitement abated, and she became sufficiently staid to attend the weekly dance. *Exactly a month after the first relapse, a second occurs*, in which she again proves boisterous, violent, and destructive; her bodily health, however, has been progressively improving since her admission. It was clearly seen that her relapses were *coincident with the menstrual periods;* but the occurrence of the next period was passed without any mental disturbance, and she was discharged, recovered, a few weeks subsequently.

Six years later, that is, when twenty-four years of age, this patient again became an inmate of our wards. She had kept well, and regularly employed as a dyer during the interval, when, upon the occurrence of a pecuniary loss by her mother, the daughter again succumbed to her inherited weakness. She was maniacally excited, though not in an extreme degree. There was marked elevation of spirits; she was well-satisfied, gay, flippant, and saucy. She talked loquaciously, very irrationally, and incoherently. Her tone is careless, almost abandoned. She states—"I am Adam's first daughter, and came here in the year 1; came here because my mother came first: I think I couldn't give you more straightforward answers, could I?" then laughs and becomes obscene and repulsive.

As on the former occasion, the excitement completely abated in a fortnight's time from admission; but only to return again in a violent form, marked by all the features above depicted, and extending over a period of about two months, when a gradual improvement succeeded, until complete convalescence was ensured.

Recurrence at the Climacteric.—Here, again, we recognise the form of insanity which is regarded as more or less characteristic at this period of life; it signifies little when the recurrent seizures originated, whether during adolescence, later adult life, or at the menopause, the symptoms of the attack existing at this latter period conform to those with which we are familiar at this revolutionary epoch. Former attacks may have been characterised by maniacal excitement—the attack at the climacteric is almost certainly one of depression, mental unrest and gloom, and of delusional perversions of the melancholic stamp. Take for instance, as illustrative of this statement, the following case in which climacteric insanity eventually issued in senile insanity:—

H. O., aged forty-eight, married. History of paternal intemperance—an aunt hung herself—an uncle died in a lunatic asylum. H. O. had been intemperate, but had enjoyed good health. Of three children borne by her, one died of convulsions. The alleged cause of her present attack was domestic anxiety, embracing supposed infidelity on the part of her husband, and occurring at the period of the climacteric. Depression of mind, lasting some six months, culminated in an attack of melancholia, with marked delusions of suspicion, from which she recovered after a residence of ten weeks.

Was re-admitted after the lapse of thirteen years. Part of this interval had been spent in Wadsley Asylum, and she had been discharged but one week when depression of spirits and apprehensive fears beset her, and she developed the delusion that chloroform was secretly administered to her. She admitted having been addicted for years to alcohol in excess; showed great defect of memory, with much blunting of intellect and emotions. Was industrious in habits, but heavy and sluggish in manner, and of vacant expression. Recovered four months after admission.

Remaining at home for nineteen months, she managed to perform her household duties fairly, and though never quite well, was orderly and manageable. Gradually, she developed delusions of the former type—suspicion of intended harm and attempts to chloroform her, basing these on tremulous conditions of her limbs, and apparently also on certain anomalous subjective sensations, perhaps hallucinatory. There was some progressive dementia and sluggishness of intellectual operations, with emotional dulness and, especially, defect of memory. Nevertheless, in two months' time she improved sufficiently to justify her discharge. Her recovery only lasted a couple of months, though she abstained from alcohol; she developed restlessness, insomnia, melancholic fears, and delusions. When re-admitted for the fourth time, she was anxious and apprehensive, but not burdened with suspicion to the same extent as before; complained of confused feeling in her head, was inappreciative and highly forgetful. From that time forth she was often restless and unsettled, fancying that her relatives were about the building, experiencing both aural hallucinations and visual illusions, and showing much mental enfeeblement and ever-increasing failure of memory. There was much pallor of the face, the skin assumed a parchment-like aspect, and the larger vessels began to evidence atheromatous change. In this state she still remains an inmate of the asylum.

The instances of recurrent insanity, recorded in eighty women, took their origin at the following respective ages:—

	Up to 20 yrs.	—30 yrs.	—40 yrs.	—50 yrs.	—60 yrs.	—70 yrs.	Doubtful.	Total.
Number of recurrent cases occurring, . . .	2	10	15	25	24	2	2	80
Number in which *first attack* occurred at each period, . . .	11	22	17	15	3	...	12	80

From this table we glean the fact that although the largest proportion of cases of recurrent insanity admitted are from forty to sixty years of age, yet the greater number of recurrents date their *first attack* from twenty to thirty years of age; and that nearly the same proportion of first attacks occur from thirty to fifty years, as for all periods below thirty. The large accumulation of cases, therefore, which appears from forty to sixty years of age, is due not to the greater tendency to the origination of this form of insanity at this epoch of life—in fact, we see the tendency decline towards fifty—but to the addition of patients who have already had attacks in earlier life. Hence, we must conclude that this epoch of life has no special influence in *originating* this form of insanity; but that it is especially prone to excite its recurrence in those who have already suffered therefrom.

Recurrence at the Senile Epoch.—The same reasoning applies to the later epoch of senility. Reverting to the same table, it is evident that although as many as twenty-four cases of recurrent insanity were admitted between the ages of fifty and sixty years, the great bulk were but relics of former storms, since three out of the number only appear to have had their first attack of insanity in this, the sixth decade of life.

J. S., aged sixty, married. Patient belonged to a highly neurotic stock. Her mother and sister were both insane; her brother cut his throat; and, at a subsequent period, her sister's son, becoming the subject of impulsive insanity, murdered his mother in the most brutal manner, kicking her to death, and causing the most terrible mutilations of her head and body. Her symptoms had shown a long premonitory stage; but four months prior to admission, she was restless, garrulous, betrayed alternations of despondency and excitability, with suspicious tendencies. When she came under observation, she was low-spirited, possessed of ill-defined apprehensions of evil, and betrayed painful emotion over trivialities, totally inadequate to provoke such distress in a normal state. She had a sharp, weazened aspect, with dark piercing eyes; was emaciated and shrunken. A decided hypochondriacal element was indicated by the prominence assigned to imaginary ailments and a craving for sympathy. She would talk for hours about her ailments, and was most importunate upon such subjects at all times. Incessantly restless, she, at times, proved most impulsive; her uncontrollable feelings being embodied by her in an imaginary ailment, "itch in the blood," to which she declared she was subject. At her worst moments, she would fly passionately at other patients without any provocation, endeavouring to inflict injury upon them, and subsequently, evince a hypocritical penitence, and querulously dwell upon her ailments; at other times, her impulsiveness tended to suicidal acts. Being discharged, "relieved," to her husband's care, she subsequently relapsed, and then attempted hanging herself in a wardrobe; but, being detected, rushed to the window with intent to leap from it. She was re-admitted, and remained at the asylum fretful, self-engrossed, importunate, and impulsive in conduct to her death at the age of seventy-two.

All our evidence, therefore, points to the **late adolescent** and **early adult life** as the period peculiarly prone to this form of nervous disease; nor need we be surprised at the fact, for at this period we meet with important revolutionary changes in the economy, the tendency to the fostering of morbid excitement, and alcoholic indulgence: at a period when in the struggle for existence the demands for a more refined, delicate, and complex adaptation are imperatively made upon the organism, and tell with especial effect, therefore, on the central co-ordinating nervous system. Given these as the influences operating in the development of the parental form, the law of "**limitation by age**" will apply as explanatory of its re-appearance at the same period in the offspring.

Recurrence in Puerperal Cases.—A certain proportion of women betray a tendency to maniacal perversions upon the accomplishment of each parturition, or during the early days of each *puerperum*. These are subjects whose heritage is probably identical with those already considered. In no particular does the seizure differ from what we know of ordinary puerperal mania, other than in this simple tendency to recur.

M. B., aged twenty-eight, married. History of paternal intemperance—a sister melancholic, but did not require asylum treatment. The patient, after her fourth confinement, a severe one, developed symptoms of insanity, and was removed to Wadsley, where she was retained about two months. Four months prior to her admission at Wakefield, her fifth confinement occurred, after which she became depressed, distrustful of her husband, and manifested distaste towards her infant; finally, threatened to commit suicide and cut the throats of her husband and family. When received into the asylum, she was convalescing from mania and rapidly became quiet, industrious, and fairly cheerful. There was neither continuance of, nor return to, the homicidal tendency; she was sent out recovered in less than two months. The interval between the preceding and the following attack was five years, during which time patient had two children; the last labour being followed by *post-partum* hæmorrhage and, more remotely, by profuse menstruation. It appeared that she soon showed mental aberration after her discharge, developing suspicious ideas about her neighbours, and fancying that they jeered at her and called her names, and it was stated that she attempted to cut her child's throat. On re-admission, there was much intellectual and emotional torpor, gloom and apprehensiveness of evil, and for some time much querulous and fretful behaviour, anxiety, and hypochondriacal fancies, which, at times, were exaggerated into actual delusions of suspicion: after a period of four months she gradually became more cheerful and composed, and one month later was fitted for discharge.

Recurrence in Traumatic Cases.—It is a fact of no small import, that 20 per cent. of the male recurrents had suffered from **cranial injury,** usually due to falls from a height upon the head, or to a violent blow causing temporary unconsciousness. The injury in no case amounted to fracture, or depression of bone, but was probably confined to molecular disturbance and nutritive anomalies thereby established. The following case is an instance of insanity engendered in an individual of the criminal type, wherein cranial injury and alcohol were important factors in the causation:—

B. L., aged twenty-seven, married, hawker; transferred from gaol. The only information as to the family history of this patient was obtainable from the latter herself, who stated that her father had been an excessive drinker, and her sister an inmate of the W. R. Asylum.

She was committed to twelve months' hard labour for assaulting and wounding a woman whom she believed to cohabit with her husband. This belief, as well as the alleged cruelty towards her of the latter, appeared to be actual facts, which, added to long and excessive indulgence in alcoholic stimulants, had produced her present mental disorder. Her forehead was deeply scarred, as the result of injuries received by a fall upon the head some years previously.

Whilst in prison, the patient behaved with great violence to her fellow-prisoners and the warders, and, on one occasion, made a desperate attempt at suicide, being discovered in her cell, black in the face from strangulation, effected by a piece of cord tied round the neck. During this time, also, she maintained an obstinate silence. She spoke on removal to the asylum, explaining that God had enjoined her to be dumb while she was in hell—*i.e.*, prison. At first abstracted and suspicious, exhibiting many purposeless tricks of gesture and countenance, she quickly became an active and industrious inmate, showing, however, some irrationality, together with considerable want of control. Although no delusions of suspicion regarding those surrounding her were manifest, yet the low type of her appearance received confirmation in occasional outbursts of violence, without adequate cause, and at all times characterised by the utmost brutality. Her behaviour, however, not deserving of the license of insanity, she was, after a residence of three months, sent back to gaol.

In less than two months she had another outbreak, and became most violent, abusive, and foul-mouthed; procuring a medicine bottle belonging to another prisoner she, with the intention of suicide, drank half its contents before she could be prevented. She was, on her return to the asylum, most maniacal, aggressive, obscene, and apparently the subject of hallucinations. In this state she remained for nearly a week, when she commenced gradually to improve, and ultimately settled down into a quite affable patient of industrious habits, with the exception of two or three passionate outbursts of short duration. Apart from actual insanity, nevertheless, she could only be regarded as of low and degraded nature. Discharged within a month of the expiration of her sentence, "relieved." Since her discharge she has frequently figured in the police courts; has been several times in prison, and, during her imprisonment, her conduct has been characterised by the utmost brutality, ferocious violence, and vindictiveness.

Frequency of Morbid Impulse.—Some 31 per cent. of the 136 instances of recurrent insanity manifested suicidal tendencies, both sexes being about equally subject to such promptings. The *melancholic* forms, and the maniacal outbursts associated with *depressing delusions*, were especially prone to such impulses; thus, six cases alone show this tendency in women below the age of forty years, all the remaining suicidal cases being at the climacteric period.

In the male, on the other hand, this morbid tendency comes out strongly in the *younger* members; but in all these instances the form of insanity was that characteristic of alcoholic and masturbatic excesses, and delusions of persecution prevailed in each. Even in the few cases occurring below forty years of age in the female, the self-destructive impulse appeared based upon congenital instability or alcoholic indulgence.

Dangerous aggressive conduct prevailed in over 52 per cent. of female recurrents, and in 64 per cent. of the males, or an average for both sexes of 58 per cent.

Hence, recurrent insanity embraces a very high proportion of individuals dangerous to others—a fact explained, in like manner, by the large number addicted to vicious habits of life, and especially alcoholic excess. With respect to this morbid impulsiveness, Dr.

Sankey, reaffirming with M. Morel its resemblance to epilepsy, writes :—"There is the same periodicity in the cases, the same impulsiveness, and the same ignorance or blindness of their own position; and though the acts of violence are not attended with any unconsciousness, yet they seem scarcely voluntary."* Amongst this class are comprised many of the criminal community of low mental type, often associated with a degraded physical conformation. These patients are almost all confirmed drunkards; spend the greater part of their life between the prison and the asylum; and, in the former, often sham insanity with the object of attaining their removal to an asylum. Here, if not repressed, they would become the tyrants of the community amongst whom they live; and, in their maniacal attacks, are most dangerously impulsive, reckless of life or limb; their conduct often prompted by the utmost brutality and the most vicious instincts. Beyond the trouble given by the criminal class in an institution where *severe* repressive measures are to be discouraged, they form a scourge to the younger and more respectable class of patients whose malady is their misfortune, and whose former associations were far different. This social evil is a blot upon our legislature that loudly calls for redress.

Hallucinations and Delusions.—Hallucinations prevailed in 22·7 per cent. of the recurrent cases—the visual and aural in about the same proportion, and both associated in a few cases; olfactory hallucinations or illusions were seldom noted, and gustatory were notably absent. Delusions occur in at least half the cases (53 per cent.). Both hallucinatory and delusional states vary with the proximate cause of the outbreak: if alcoholic excess enters largely into the causation, we may anticipate associated ideas of self-importance, rank, power, wealth, and suspicion of perfidy upon the part of those around him. One patient receives a nightly visit from his satanic majesty; another sees imps around him, hears voices beneath the floor—the noise and rumble of machinery, which his morbid imagination frames into some idea of coming torture. Another patient, twenty-eight years of age, addicted to intemperance in drink, and the subject of a serious cranial injury in youth, calls himself Sir Roger Tichborne, and accuses his relatives of filling his bedroom with the vapour of chloroform. Another young alcoholic subject owns property "to the value of thousands a year"—has extraordinary muscular power, and can "walk eighty miles a day continuously," Delusions of poisoning are frequent in these alcoholic cases, as are also notions of being deprived of property and rights, or being pursued by the messengers of the law. One typical case, aged thirty-eight years, with a history of paternal intemperance and strong collateral insanity (two sisters being insane), himself for years a heavy drinker, developed, upon his third outbreak of insanity, the notion (from certain subjective feelings referred to the chest) that some mysterious clockwork was concealed there, which caused him much agony and deranged his mind. He could scarcely be restrained from injuring himself, and often begged to be operated upon with a view to its removal. He frequently bruised himself seriously over the front of the chest by violent blows of the closed fist.

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* *Op. cit.*, p. 175.

J.B., aged thirty-six, married, a fish-hawker. Has had no previous attack of insanity. Eight days previously he became maniacal and dangerously aggressive; was under the constant charge of two men at the workhouse, whither he had been taken. There he attempted to leap from a window, and struck his forehead with a soda-water bottle, inflicting a severe gash, with suicidal intent. Upon admission he was depressed, heavy, and sluggish, yet sufficiently calm to give a clear account of himself. He was ill nourished, with flabby muscles; complexion sallow and dirty; several deeply-incised wounds recently inflicted were observed on forehead; expression depressed and torpid. Patient gave a history of excessive drinking for some years past, as also of an attack of *delirium tremens;* had been drinking quite recently, and "saw all sorts of things about him." Two sisters were insane, but all other antecedents free from neuroses; his father was a heavy drinker. He admitted being jealous of his wife, whose fidelity he questioned, and he had therefore deserted her and his children. During the first week he remained morbidly depressed, pensive, self-absorbed, and inactive; rarely spoke, but said he was quite "beside himself" when he cut his forehead; appetite good; "nasty foul objects" surround him; was gloomy. A week later no further illusory or hallucinatory state prevailed; appeared of normal consciousness, and was shortly afterwards employed for a time at work, and discharged.

The next occasion upon which we hear of him was two years following his discharge. During the interval he had gone to America, resumed his drinking habits, and was soon an inmate of the Trenton Asylum. When re-admitted here, he had developed typical delusions; declared that some clockwork was within his chest—the movements were incessant, caused him great suffering, and allowed him no rest; felt impelled to commit suicide. The voice of a man also was heard speaking to him from within his body. He was irritable, violent, dangerously suicidal, and his language abusive and blasphemous. He describes the "clock" in his chest as causing sensations like "a chopping machine;" it feels as if it would "rive his heart out;" begs to have a surgical operation performed upon his chest, that "the machinery may be cut out;" and he points to several recent bruises over the manubrium inflicted by his clenched fist to relieve the anguish he feels. Believes certain men have placed the "clockwork" there to make him jump and dance about whenever they choose. He became much agitated and excited during this narration; talked hurriedly and incoherently.

For two months he continued to exhibit the symptoms above described, was always excitable, spoke in a hurried flow of words, but was coherent; he proved friendly in his disposition to those around him, took much interest in his domestic employments, and was fairly cheerful.

A relapse occurred two months later; the delusion became again more prominent, and he repeatedly threatened to cut his throat. For the subjective anomalies leeches were applied to the manubrium, but without result. Bromide and chloral were then given with very considerable benefit; the pain gradually subsided; his sleep was ensured at night; and in six weeks' time he declared himself free from any morbid sensations, had lost his delusional notions, was quite rational in converse, and left the asylum.

In one month later he again became an inmate, suffering from his fourth attack of mania-à-potu. The "clockwork" still drives him frantic; he must cut open his own chest and remove it, or will "split open the chest of some one else;" he will "murder those who persecute him." Says he went home, worked steadily, and remained quite well, abstaining from drink for two weeks, and then the terrible feelings in his chest began again, causing sharp lancinating pains, which had "the power of arresting his breathing, and caused agony even to his finger-tips." He strikes himself violently upon the chest in sheer despair, and is much bruised over the sternum. Threatens to take his own life unless relieved by some operation; is very excitable, garrulous, circumlocutory, irate. He took liquor opii with spiritus ætheris sulph. (āā mins. xv.) twice daily. During the first fortnight he derived relief from the opiate, but continued to exhibit mild maniacal excitement. He remained very deluded, and about this time, having secured a knife he retired to a closet, and inflicted a deep incised wound down the front of the chest over the sternum ere he was detected. Chloral and bromide (āā grs. xxx.) were substituted for the opiate twice daily; steady improvement took place—he lost his painful sensations, but upon retrospect he still affirmed the reality of the diabolical machinery which had been introduced into his chest. A few weeks later he was finally discharged as recovered.

Prognosis.—A large proportion of the recurrent insane who enter our asylums after successive attacks of insanity become chronic inmates, or are discharged as partial cures only, or the disease proves fatal. Considerable disparity, however, appears to be maintained between the rate of recovery amongst the male and the female residents; the former range as high as 71·4 per cent. of the total number, the latter 57·5 per cent. If we group together as "unfavourable cases" all partial recoveries, deaths, and chronic remnants of the recurrent female class, we find these amount to 40 out of a total of 100 cases.

We find that of the "relieved," the deaths, and the "remaining," only 10 cases of the 40 were under forty years of age, the melancholic form which prevails beyond this age being a far less recoverable form of insanity than the acute excitement of earlier life. One most unfavourable aspect of recurrent insanity, therefore, is that of a recurrence at the period of forty and upwards; in fact, if we fail to break through the periodicity established in our patients' morbid tendencies, before this age, the outlook is very ominous—the lucid intervals between their attacks become of shorter duration, and the mental stability at their best moments so insecure, that in *impulsive* forms it becomes imperative to keep them under continuous supervision. The period of calm between the attacks is not only more uncertain in its duration, but there is now betrayed a steadily advancing mental enfeeblement; and, as dementia deepens, so do the attacks of excitement or depression become more frequent and more prolonged; yet, even in these advanced cases, the periodicity of the disease is maintained. Dr. Blandford cites the case of a man of more than eighty years of age, who came under his observation, whose first attack happened when he was seventeen, and who had been placed under supervision for recurrent attacks three-and-thirty times.* A patient at the West Riding Asylum between the age of thirty-two and forty-two had an attack almost every year, nine in fact; between each of which she was discharged, and resumed her household duties with energy and ability, manifesting no intellectual impairment or emotional instability.

In another case—a married woman, addicted to heavy drinking, had her first attack at the age of thirty-five, and from this period through the whole of the climacteric, was subject to repeated attacks of maniacal excitement of a wild, boisterous, and dangerous nature, with obscene and most objectionable behaviour. In her case, asylum supervision was required on ten occasions up to the age of sixty years; but, for several other attacks of excitement, she was treated at home and recovered.

In yet another instance, a young girl of congenitally weak moral control suffered from three successive attacks, with complete lucid intervals, between the ages of seventeen and nineteen; she returned home and resumed her duties in the intervals of her attacks. Three further attacks occurred up to the age of twenty-four years, when her mental equilibrium was so far unsteadied that she continues, up to the present time (a period of ten years), an inmate of our wards. During this latter residence she has had repeated attacks of excitement, and her periods of calm are now greatly broken by hysteric symptoms, in which erotic manifestations are prominent. In her case, also, diurnal calm often alternates with nocturnal restlessness, gentle excitement, and garrulity.

> "The disorder, once set up in the individual's constitution, is prone to recur, and we must examine the whole question of the periodicity of disease, as well as the conditions of the first attack, before we can hope to throw any light upon the subject. This much we may conclude, that the conditions which precede the first are not necessary to subsequent attacks; that as epileptic seizures may continue after the ostensible cause of the first fit is removed—*e.g.*, worms—so the disorder once recurring may repeat itself, persistently remaining as a vice of the constitution of the individual, of which it now forms a portion" (*Blandford*)†.

With this statement of Dr. Blandford we fully agree, so long as it is understood that the conditions of the first attack, to which he alludes, are *environing*, and not organised, conditions; for it is all-important to bear in mind that a very large proportion of the cases exhibit a powerful hereditary predisposition to insanity, and that we always fail to elicit to the full extent from the most careful enquiries the magnitude and importance of inherited neurotic conditions. Yet out of the 136 persons who were subject to recurrent seizures, permitting them to return to their homes in the intervals of their attacks, we found very definite and undoubted evidence of inherited insanity, of other neuroses, or a history of parental intemperance, or of severe cranial injury in seventy individuals (51·4 per cent.). If, therefore, in *one-half* the cases such powerful predisposing factors be found, any one familiar with statistical research in this direction will add a wide margin for similar agencies in other cases not divulged, too remote for detection, or in patients whose antecedents are utterly unknown, as so often occurs in the class with which we deal at large pauper institutions. If we now group together promiscuously all the recurrent individuals of our past ten years' experience at the West Riding Asylum, we may construct from their histories a chart of recoveries, such as is given on Chart A.

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* *Insanity and its Treatment*, p. 71.

† *Op. cit.*, p. 72.

A steady increase occurs in the number of recoveries up to the sixth month, when a climax is reached—fifty-four of the total 105 cases of recovery (or 167 total cases under treatment) having been discharged. One-half the recoverable cases, therefore, are well by the sixth month; a notable fall occurs between the seventh and eighth months, with a slight rise of seven cases at the ninth month, again to decline to the level of one or two cases monthly until the thirteenth month, after which the recoveries are few and distant—*e.g.*, one at twenty months, one at two years, and one at six years.

The recovery line for the men differs from that of the women in attaining the climax two months later; the largest proportion of cures for female recurrents (seventeen) taking place during the fourth month of their attack, and steadily declining to the seventh; whilst the maximum of male recoveries (twenty-six) occurs at the sixth month, dropping suddenly to one case for the seventh and eighth months. Of the females, whose recovery was protracted beyond the ninth month, all without exception were *above forty years of age*, had suffered from several previous attacks, or were subjects of *congenital mental defect.*

Circular Insanity (Folie Circulaire, Folie à Double Forme).—This is a condition where melancholia and mania, or the reverse, follow each other in invariable sequence, with or without a shorter or longer interval of lucidity. Between the attacks of melancholia and mania there may exist a period when the subject can be regarded as in neither phase, but calm, lucid, and apparently stable; so that there may be some danger of mistaking the lucid interval as one of actual recovery, rather than the transition period from one to the other mental phase.

Or again, there may be no such intermission, but the depression may gradually diminish, passing insensibly into excitement.

In like manner, the successive cycles of excitement and depression

CHART of RECOVERIES.

INSANITY AT THE CLIMACTERIC.

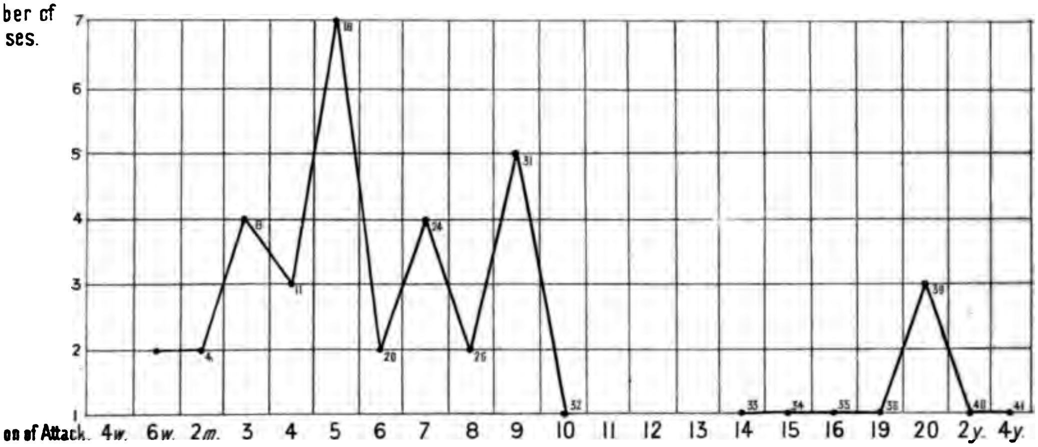
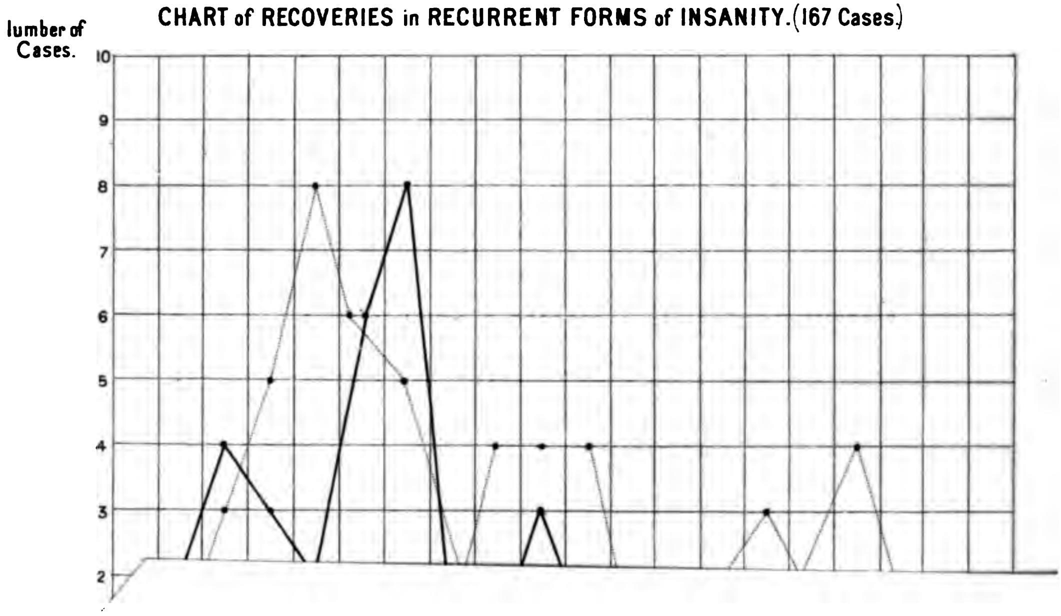


CHART of RECOVERIES in RECURRENT FORMS of INSANITY. (167 Cases.)



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may be separated by so short an interval of lucidity that the attacks themselves appear continuous—no intermission whatever occurring. We therefore recognise in the history of such cases the stage of

(*a*) Maniacal reductions : (*c*) Melancholic reductions :
(*b*) Transition : (*d*) Interparoxysmal lucidity.

In many cases the stages *b* and *d* may be entirely absent.

Should there be no interparoxysmal period the attacks of melancholia and mania alternate constantly, establishing thus a continued insanity of alternating mental phases—the *true* Folie Circulaire of many authors; whilst, if the interparoxysmal stage be well marked, we have the *intermittent form* of circular insanity.

The stage of depression may reproduce *any of the usual forms of* melancholia, stupor, or melancholic stupor; in no respect is the form of reduction *peculiar* to this disease; in like manner, the maniacal reductions may extend to any depth, issuing in the gentlest or the most acute degree of excitement. In the nature of the depression or excitement there is no feature which specially stamps this affection beyond the alternating character of the reductions.

Although in individual cases each attack is almost an exact counterpart of that which has preceded it—and this as regards *intensity* and *nature* of symptoms, *duration* of each stage and of the complete cycle—yet, in different subjects, great diversity is shown in all these particulars. Each stage of reduction may last from a day or two to several weeks or months, and the interparoxysmal period is subject to the same uncertainty. The affection is notably a chronic and incurable one; and, although in certain cases a year or more may intervene betwixt the attacks, the tendency to recur soon betrays itself, and very rare indeed are the instances of recovery.

Etiology.—Although classed under the heading of recurrent forms of insanity, it will be seen that the family alliance is by no means so close as this might at first seem to imply. The tendency to recur; the reproduction of similar mental phases; the peculiarly incurable nature of the disease; and lastly, the strong hereditary basis upon which it is engrafted, might suggest this alliance. When, however, we more closely study the etiology of this affection we are at once struck by much dissimilarity; the following factors illustrate this:—

Sex.—It prevails far more frequently in woman than in man; all writers alike assert the influence of sex in this direction.

Age.—Puberty and the period following, up to thirty, afford by far the greater proportion of cases of Folie Circulaire.

Heredity.—Direct heredity plays an important *rôle* in its development. A neurotic heritage, epilepsy, alcoholism, chorea, hysteria, and insanity are the constant antecedents of this affection in the ancestry.

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Treatment.—When considering the etiology of recurrent forms of insanity, we emphasised the ***hereditariness*** of the affection, and the unstable, defective, mental organisation of the subject. We regard the indulgence in alcoholic stimulants as having, perhaps, a more fatal effect upon the subjects of this, than of those of any other form of insanity. Alcoholic treatment here is decidedly most pernicious. In fact, all forms of the explosive neuroses do better without any alcohol—even when their disease does not appear to have been engendered by undue indulgence in stimulants. We often find that the subject craves for alcohol, and also for all sorts of mental excitation; but these must be withheld wherever they tend to induce the least emotional instability. Our sheet-anchor in the treatment of these affections is much outdoor exercise, with active manual employment for both sexes, long walks, cheerful society, and avoidance of association with the more excitable chronic lunatics. With this there should be given a liberal, wholesome dietary. By some authorities it has been considered well to limit the meat-diet, and to add largely to the farinaceous and vegetable constituents of the food—a suggestion which applies also to the epileptic and other convulsive neuroses. We do not ourselves regard the question of the advisability of a farinaceous diet as conclusively proved in the case of the convulsive neuroses; the most important attempt to practically test the question in epilepsy, was recorded by Dr. Merson, in the "West Riding Asylum Medical Reports for 1875," the result being in the main favourable to this dietary, but based upon too limited a number of instances to warrant final acceptance.

Bromide of potassium, in combination with Indian hemp (30 grains of the former to a fluid drachm of the tincture), is the best remedy for the states of acute excitement. The patient's appetite is never prejudicially affected by it. In most cases of this class they take food more readily with, than without, this treatment.

The exaltation of the sexual instincts, which so often characterises these recurrent seizures, renders iron and the compound phosphates inadmissible in many cases. In most adolescents the recurrent attacks are best met by bromides alone, careful attention to the bowels, regular exercise, the spinal douche, followed by friction of the surface; and, if there be much insomnia, an occasional chloral draught.

The phosphatic preparations, with cod-liver oil, may, however, be given with benefit in the recurrent attacks of melancholia incident to the climacteric, and the bromide of potassium, in combination with the perchloride of iron is often advantageously prescribed.

In the circular form of insanity the general principles which guide us in the treatment of mania and melancholia must also here be relied upon. Attempts have been made to cut short the periodicity of this affection by such remedies as quinine, digitalis, cannabis, morphia, and the bromides. Quinine has been administered in large doses, up to 30 grains, but with indifferent results; a better palliative treatment is that of the tonic regimen applicable to all cases of periodical forms of insanity.

EPILEPTIC INSANITY.

Contents:—Definition—Epileptic Neurosis—Immediate and Remote Results of Epileptic Discharge—Diffusion-currents—Nascent Nerve-tracts—Discharge from Sensory Areas—The Aura in Sensory Epilepsies—Epileptic Amaurosis, Hemianopsia and Hemianæsthesia—Champing Movements—Pre-paroxysmal Stage—Premonitory Stage—Special Sense Auræ—Vaso-motor and Visceral Auræ—The Epileptic Paroxysm—Grand and Petit Mal—Post-paroxysmal Period—Post-epileptic Automatism—Case of E. C.—Status Epilepticus—Inter-paroxysmal Stage—Epileptic Hypochondriasis, Automatism and Impulsiveness—Medico-legal Relationships—Impulse—Delusion—Malingering—Reg. *v.* Taylor—Treatment of Epileptic Insanity.

By **epileptic insanity** we mean that form of mental derangement in the antecedent history, oncome, and further development of which we recognise an intimate connection with the epileptic neurosis. Such functional disturbances of the nervous mechanism as issue in what are termed epilepsies may, or may not, have for their accompaniment serious mental derangement. Epileptic fits may continue for years with slight, or scarcely appreciable, mental disturbance. If, however, the epileptic neurosis presents on the physical side a parallel disorder of mind, we speak of it as epileptic insanity. Epilepsies may develop during the course of other cerebral diseases associated with insanity as pure accidents, or as an intercurrent affection, in chronic disorganisation of the brain, in softening from embolism or thrombosis, in senile atrophy and decay, during the progress of general paralysis of the insane, or in certain cases of chronic insanity, the epilepsy then being merely an accidental complication of the primary affection, and dependent, probably, in part upon the direction taken by the disease. Nor can it be questioned that epilepsy may arise as an independent and intercurrent disturbance in subjects mentally afflicted, having no direct connection with the primary cerebral derangement, and it becomes therefore imperative that we learn to recognise such morbid lineaments, so to speak, in the mental affection as indicate its kinship to an **epileptic neurosis.**

The mental derangement of the epileptic may assume the form of maniacal excitement, of melancholic depression, of mental enfeeblement or dementia, or of delusional perversion or perversions of the moral being: any one or more of these states may be revealed by the patient. As in all cases of insanity alike, so epileptic insanity notably presents periods of heightened functional commotion, with intervals of comparative calm, periods of sudden and excessive, though transient, dissolutions, and the persistent impairment due to a constantly advancing, though gradual, dissolution.

The *immediate* results of an epileptic discharge are seen in the deep reductions of epileptic mania—a transient condition only; the *ultimate* results of repeated attacks in the gradual and persistent impairment of the mental faculties seen in epileptic dementia. Hence, in studying epileptic insanity, we have to consider the acute symptoms or immediate after-effects of a fit, as well as the chronic impairment presented during the intervening periods between the attacks. It is not, as before stated, *every* form of epilepsy which is prone to issue in mental derangement, if by epilepsy we mean what Dr. Hughlings-Jackson means; that is—"A sudden, rapid, excessive, occasional, and local discharge of cerebral cortex."*

It is when the functional disturbance occurs in the highest nervous arrangements of the cerebral cortex ("the substrata of consciousness") that the mind is prone to suffer. An all-important principle which the same authority has taught us to recognise is that, in these epileptic seizures, there is a *brutal* expenditure of force wholly out of proportion to the normal physiological outlay, and wholly inconsistent with continued healthy activity of the parts concerned. So severe is the explosive violence, that the nervous tracts traversed by the storm are so damaged as to be rendered transiently incapacitated for the further conduction of the nerve current, and the centre itself is paralysed for the time by its enormous expenditure of energy.

If we attend carefully to this fact, it will be apparent to us that the transient paralysis of the motor centres and nerves is not the only or most important sequel of the seizure. We are aware that every vivid mental rehearsal initiatory of an act (especially when the action is itself suppressed) is attended by diffusion currents, which, according to the physiological law of least resistance, affect first the smallest musculature, *e.g.*, the eyeball and facial muscles; and even when these results are not apparent, expend themselves along intra-cerebral tracts, arousing sensory excitations and correlated feelings. Just as the substrata of these representative states affect cortical realms other than those in which the primary excitation arises, so likewise, during the accomplishment of every volition, the act is accompanied by so-called associated actions (*e.g.*, the associated contraction of pupil and convergence of eyeball), and this series of associated movements is a very large one in the active manifestations of the organism. But this is not all. We know that both the initiatory energising of the cortex and its eventual actualisation are attended by numerous complex feelings, such as a memory of similar acts previously performed, of their results, and of the notion of the utility of the act to the "individual's" welfare.

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* See *West Riding Asylum Reports*, vol. iii., p. 331.

All this means, of course, diffusion currents around the primary discharging centre.

If, then, all energising and discharging of motor centres be accompanied by such effects in related centres, how much greater will be the effect when the motor areas are overflooded by the brutal explosiveness of epileptic discharges. We shall then have not only the paralysis of the conducting tracts, but also a dangerous flooding of those delicate, yet indefinitely extended, tracts of intra-cortical nerve-tissue, upon which the very evolution of the nervous structure depends. If we place any credence whatsoever in the theory of nerve-genesis so elaborately worked out by Herbert Spencer, we must regard this undue **forcing of nascent nerve-tracts,** as yet incomplete in their formation, as a most serious matter in epilepsy.

It is important to observe that any one part of the cerebral cortex may be the site of an epileptic discharge; and hence, the resultant phenomena will be co-extensive with the multiplicity of cerebral functions, and as varied in their nature as *they* are varied. Discharges in motor realms will thus afford endlessly diversified combinations and sequences of spasms, whilst discharges from sensory realms will likewise implicate correspondingly complex centres. The former are open to *objective* study; the latter, as being purely *subjective*, can only be gleaned by information given us by the patient. Again, local discharges initiated in motor realms may spread to other motor areas, or, from being of hemispheric origin, may become bilateral in their distribution, or, spreading backwards into sensory realms and the highest and most complex of centres, issue in loss of consciousness. As regards this implication of consciousness, "all depends on the momentum of the discharge, and, therefore, on how far it spreads" (*Hughlings-Jackson*).* In like manner may arise pure sensory epilepsies, with, or without, loss of consciousness in the full acceptation of the phrase; or a primary sensory epilepsy may spread into more purely motor realms, and issue in general convulsion, the discharge, as it were, being reflected on to the motor sphere. We thus see how infinitely varied may be the resultant of epileptic discharge from any unstable area of cortex. In unilaterally commencing convulsions—viz., those due to local discharges in one hemisphere—we can usually trace the spread of the discharge with facility; this is, however, not the case with the epileptic seizures associated with insanity. Here we more frequently observe attacks of **petit mal,** or else that form of **grand mal,** in which the loss of consciousness is early and complete, and the spread of the discharge so rapidly general that the whole body is almost simultaneously affected by the convulsion. This rapid rundown of mechanism, comparable to the rupture of the mainspring of a watch, renders it impossible in most cases to distinguish any sequence in the resulting spasms. It is truly a *universal spasm*, or, to use Dr. Hughlings-Jackson's vigorous phrase, "a clotted mass of movements." In like manner consciousness is then lost at so early a period, and this so suddenly that the patient falls instantly, as though struck senseless by a blow.

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* *Loc. cit.*, p. 268.

The discharge from *sensory* areas cannot, from the very nature of the case, be followed; we can only learn the existence of an aura by the subsequent statements of the patient; but—impressed only by the results of the motor discharges—we must not lose sight of the fact, that equally powerful discharges, of which we see no result, may pass along sensory areas at the period when consciousness is abolished. Undoubtedly the muscular spasms are likely to attract to themselves undue attention on the student's part; and he forgets for the time that still more noxious effects are being produced in the areas of mental and sensorial activity silently and concealed from his view. It is, therefore, all the more important that one should keep these unseen results in mind, and watch carefully for such evidence as may arise of the implication of the sensory portion of the brain, for such evidence is forthcoming at certain stages of the affection.

It is very obvious that amongst a large number of epileptics in our asylums who suffer from mental derangement, there is very great divergence in the history and progress of their affection—the phenomena of their disease by no means present a dead uniform level. Some remain inmates for very many years with the intellect but little impaired, and then only at those periods when they become subjects of epileptic seizures; others (with few, if any, convulsive attacks) betray at long intervals periods of depression, of moroseness, or of excitement, during which they are more or less irresponsible for their actions; yet, in the interim, they are perfectly rational, cheerful, amiable, considerate for others, and obliging. In others, again, the mind becomes rapidly enfeebled; and during the period of their "fits" the reductions are so profound that absolute dementia and stupor, or perhaps wild ungovernable fury, prevail. And, yet again, with but little essential difference in the motor disturbance of two cases, the wreck of mind in the one may stand out in strange contrast with the clearness of intellect in the other. The resulting dementia therefore (so-called "effects of the fits") varies very considerably in degree, so that each individual case may be unlike the others in this respect; and this is undoubtedly dependent upon the varying seat of the primary discharge from the cortex. To quote Dr. Hughlings-Jackson—"From this it follows that there is, scientifically speaking, no entity to be called epilepsy; but innumerable different epilepsies as there are innumerable seats of discharging lesions. And as the first symptoms in the paroxysm is the first effect of the discharge of the centre unstable, any two paroxysms beginning differently will differ throughout, however little."* From all this it becomes sufficiently obvious that we must not rest satisfied with a mere observation of the motor discharge exemplified by the convulsive seizure; but, we must likewise question our patient closely upon his sensations and mental disturbance immediately preceding the loss of consciousness, and observe closely his condition as presented after the paroxysm and up to the full re-establishment of conscious activity.

What are some of these indications of discharges in sensory realms? A patient at the West Riding Asylum after each severe attack of fits becomes completely blind, and gropes about on hands and knees—epileptic amaurosis is, however, an infrequent effect of this disease.

Another epileptic becomes hemianæsthetic on the left side of the body after certain convulsive seizures; and this anæsthesia is attended with a corresponding state of the retinal fields; there is left homonymous hemianopsia, associated also with impairment of the other special senses of the same side. Indications of discharges in sensory realms are afforded during this stage of re-energising, by the champing movements of the jaws, with corresponding movements of the tongue, probably indicative, as has been stated by Ferrier, of discharges from the centre for taste, the movements being thus reflexly induced. The rubbing of the hands together—the attention of the patient evidently being attracted thereto—probably means that morbid sensations are referred to those parts. A very frequent action amongst such patients is the rubbing of the open hand upon the knees, or the slapping of their thighs with the palm of the hand. We must, however, be careful to avoid arriving prematurely at a decision, that this is due to discharges in sensory realms, initiating the movement; it certainly may be due to a more complex mental state. Thus one intelligent patient explained this action, of which he seemed conscious, by saying, that he did it because he thought he could "bring himself by this means more rapidly *out* of the fit." Discharges from the substrata of visual sensori-motor areas of the cortex will often be indicated in fantastic movements of the hands, as though the patient were disentangling imaginary skeins of thread in the air. Other subjects, and these are by no means infrequent, appear to be following imaginary objects on the floor, or peer in some one direction; or, again, scrutinise with incessant vigilance the floor and furniture around them, as though searching for some lost object. With all this there may be considerable motor automatism; the subject may climb upon tables or the window-sills still searching apparently for some object; or he may remove his coat, turn out the contents of his pocket, &c.

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[*] *Loc. cit.*, p. 270.

One of our patients invariably after his fit empties his pockets on the table, secures his pipe, and placing it (although empty) in his mouth, marches to and fro with a self-satisfied look and contented mien.

All cases of epileptic insanity should be rigidly studied with a view to (1) eliciting the condition of mind immediately preceding an attack; (2) the essential features of the epileptic seizures; (3) the subsequent period of reinstatement of consciousness; (4) and lastly, the mental state prevailing in the period intervening between the "fits." We, therefore, divide our remarks under the headings of—Firstly, the preparoxysmal stage; secondly, a premonitory stage (often absent); thirdly, the paroxysmal stage; fourthly, a post-paroxysmal stage; fifthly, the interparoxysmal period.

1. The Preparoxysmal Stage.—The epileptic insane are especially prone to exhibit indications of an approaching seizure; nor is this surprising, when we recall how slight departures from the normal state of healthy cerebral nutrition betray themselves in all our subjective feelings and moods. The grave nutritional anomalies upon which an explosive neurosis depends might well be expected to declare its advent thus—subject, of course, to the special site of nutritional derangement. A change in character is thus frequently recognised during a period of hours or even days antecedent to a seizure. An able and intelligent attendant will so study his cases, that he at once detects the little minor changes in the patient's disposition, indulges his whims, endeavours to sooth his morbid irritability, and especially guards the subject at this period from unnecessary annoyances. Thus, we frequently hear excuse made for some patient's lapses of temper, or unseemly behaviour, "Oh, he's just going to have his fits, sir, he will then be all right." In asylum life, amongst the intelligent class of nurses, the fact is universally recognised that a premonitory stage of great irritability is often seen, and the effect of a convulsive attack will be to clear up the mental atmosphere.

The mental disturbance thus preceding the epileptic paroxysm presents very variable features—(1) melancholic gloom and despondency may prevail; (2) hypochondriacal perversions, which may have been persistent during the patient's interparoxysmal stage, may now become exaggerated and intensified; (3) restless, objectless wandering may indicate the uneasy discontented mind; and the subject may complain of this unrest, of being unable to follow his usual occupation, incapable of keeping his mind upon any subject long together; he cannot read; his sleep fails him; he becomes indifferent to his meals and inattentive to his wants generally; (4) a *vague* dread of impending evil is occasionally expressed; but this is more frequent as a genuine *aura*—a psychical state, the immediate accompaniment of the commencing epileptic discharge; (5) joyous elation may precede an attack, a general state of optimism be present, often associated, however, with gross egoistical sentiments; (6) confusion of ideas, diminished vigour of attention and memory are also peculiar to this stage; this is the first symptom, for instance, betrayed by an epileptic compositor employed at this asylum; with him there is also at this time a notable degree of irritability and irrepressible garrulity; (7) delusions of suspicion are a prominent feature before epileptic seizures in certain of the insane, and may form the incitants to acts of dangerous or homicidal violence.

An inmate of the West Riding Asylum almost invariably betrays to his attendant this state of mind; he stalks up and down the wards, assumes a defiant attitude and bearing to all around, keeps a vigilant eye upon each passer-by; and occasionally beckoning the attendant, reveals to him privately the existence of an imaginary conspiracy to poison him. This patient wholly ignores the fact that he has fits—"Oh, they say I take fits, you know" (with an incredulous smile) "but, I know what ails me;" and then with a mysterious air—"What they put in my food and medicine explains everything." At times he openly accuses the doctors of drugging his food and drink, *and always in the stage preceding convulsion.* In this preparoxysmal stage, depression always prevails; the convulsive seizure occurs, and as the attendant emphatically and truthfully asserts—"he is then a new man." This patient also believes he can ward off the effects of the poison by drinking his own urine, which he has been detected doing on more than one occasion, and with this avowed object in view.

2. Premonitory Stage.—This is not truly a stage, but the first period of the paroxysm itself; yet it is convenient to consider it separately in accordance with the old notion of the phenomena, the so-called warnings or **auræ.** In fact, the phenomena embraced by this period, the epileptic aura, are but the subjective aspects of the nervous discharges in cortical centres, the initiatory symptoms preceding that loss of consciousness which leaves the remainder of the convulsive paroxysm *minus* a positive mental counterpart. For it must be remembered that in all these excessive discharges along highly specialised sensori-motor mechanisms the subjective phases are of but transient duration, only during the earliest period of the attack; the objective are obtrusively present, but from the early failure of consciousness have no mental correlates. Since, however, the epileptic aura constitutes the *earliest* symptom of the *actual discharge*, when correlated with the physical accompaniments of the attack, it facilitates our comprehension of cerebral activity, and the parallel series of psychical manifestations. To the student of psychology it is of intense interest, and should be studied with the greatest care.

And, in the first place, since any of the regions of the cerebral cortex (which are the anatomical substrata for all forms of conscious activity) may be the site whence an epileptic discharge originates, so the phenomena of an *aura* may be co-extensive with all forms of sensation whatsoever, and may even be constituted by more elaborate forms of psychical activity. The sensation constituting an aura is, therefore, referred to any portion of the environment, including in the latter term the body and its organs generally. We thus may get *auræ* of—(1) the special senses; (2) of the visceral or organic sensations; and (3) intellectual or psychical auræ. A few remarks on these sensations will render the subject clearer.

(*a*) **Special-Sense Auræ.**—It must be remembered that in the five special senses—taste, smell, touch, hearing, and sight—we have an ascending scale of sensations entering more and more intimately into connection with our intellectual life. **Taste** and **smell** have the least intellectual element, and are specially characterised by their slight recoverability in idea—*i.e.*, in persistence or capability of being recalled in the absence of the object; although both are capable of much improveability by education (*Bain*). On the other hand, **touch** is a much more intellectual sense, highly discriminative as to locality, and capable, in conjunction with other senses (and especially the muscular sense), of giving us ideas of the form, dimensions, and position of objects in general; its essential intellectual factor is dependent on the conjoint agency of the muscular sense (*Bain*). **Hearing** and **sight** attain the maximum as regards intellectuality; are highly co-operative; exquisitely discriminative in their powers; highly persistent and recoverable; as well as capable of almost unlimited education. **Sight** is, of all the senses, pre-eminently characterised by the faculty of objectivising, and in fact enters much the most largely as an ingredient into the constitution of object-consciousness. On the other hand, at the further end of the scale, the sense of taste and of smell (and still more notably the sensations of organic life) are characterised by their inherent subjectivity, or the greater difficulty experienced by us in discriminating between subject and object. The drift of these remarks will be at once apparent to the student when he considers that the least discriminating and most subjective of these series of sensations (the organic or visceral and taste and smell) have least connection with the intellectual operations of the mind; the most discriminating and most objectivising (the high or special senses) have intimate connection with the intellectual operations, and that, *therefore*, auræ, consisting of the former sensations, must be referred to the implication of the substrata of the crude sensations of organic life, and the emotions—those of the latter—to the substrata of the highest activities of the mind, although they also enter into the emotional life of the being.

Organic Sensations.—The innumerable impressions which must arise momentarily and co-instantaneously throughout the organism during the healthy activity of all its tissues, its muscles, bony framework, viscera, and vascular apparatus form in their aggregate what are termed the **sensations of organic life.** Many of these, such as the visceral and vascular, have phases of "unfelt" sensations, or, at least, sensations not discriminated from the vast mass of sensations created by the functional activity of the body at large, with periods of emphatic expression—*e.g.*, hunger and thirst. The "unfelt" sensations, however, rise into prominence in morbid states of the system, and we then get those intensified organic sensations, which cause much discomfort and contrast with the normal massive feeling of *bien-être*. In the epileptic, likewise, we get such sensations aroused in the organic auræ; they are distinguished by the massive and all-pervading character of the sensation. We may take Professor Bain's classification * as embracing these sensations of organic life, which are thus liable to derangement:—

SENSATIONS ARISING FROM

(1) Muscles.
(2) Bones and ligaments.
(3) Nerves and nerve centres.
(4) Organs of circulation.
(5) ,, respiration.
(6) ,, digestion.

To which we may add those of the urino-genitory apparatus.

Visual Auræ.—These occasionally precede the seizure in epileptic insanity, but, as pointed out years since by Sir J. Crichton-Browne, auræ are not of frequent occurrence amongst this class of epileptics. † When they do occur, the visual auræ consist usually of crude sensations, balls of fire, coloured light, glittering sparks, &c.; thus *G.M.* sees a number of sparkling stars before his eyes, all around "looks dim;" and if he holds a book in his hand, ere it falls "the letters all run into one another." *W.B.* has a warning described as a doubling of objects around him, as if by "cross sight," meaning that he supposed it was due to a transient squint. Red and blue are the colours more frequently seen in these visual auræ (*Gowers*).

Auditory Warnings.—These are less frequent than the visual; but are occasionally met with in elaborate form. Dr. Ross speaks of hissing, singing, or explosive noises; of a noise in the ears, followed by vocal utterances, in some cases of ordinary epilepsy. ‡ Dr. Gowers speaks of a crash, a whizz, a hiss, or whistle; or on the other hand, a loss of hearing, strange stillness preceding the loss of consciousness. These are rarely recognised in asylum epileptics. Consciousness, as a rule, is in them too early lost for these phenomena to occur.

Gustatory and Olfactory Auræ.—These are the least frequent forms of auræ met with in epileptic insanity. In the patient *J.V.* the convulsive seizures were invariably preceded by such affections of the sense of taste as would justify us in regarding them as gustatory auræ. The intimate connection between the sense of taste and that of smell renders the differentiation between hallucinations of these senses dubious, and at times impracticable. We must carefully exclude the instances of perverted sensibility which so frequently engender sense illusions in the epileptic subject, giving rise to delusions of being fed upon human flesh, or similar revolting notions.

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* *The Senses and Intellect*, Alexander Bain—"Sensations of Organic Life."

† *West Riding Asylum Medical Reports*, vol. iii., p. 160.

‡ *Diseases of the Nervous System*, vol. ii., p. 919.

Vaso-Motor Auræ.—This form is exemplified in the case of a patient whose fits are always preceded by unilateral vaso-motor disturbance—marked mottling of the skin of the palm, associated with morbid sensations; the patient invariably opens the hand and inspects it critically, turning it over and over again, and feeling the skin with the fingers of the other hand. Consciousness is then lost, and the arm so affected becomes convulsed.

(*b*) **Visceral or Organic Auræ.**—These are the more prevalent sensations recognised in epilepsy, as stated by Sir J. Crichton-Browne.* The feeling is one of weight at the epigastrium, or a fulness or distention of this region. This feeling often rises to the throat, causing a sense of great discomfort—the patient beginning to pull at his collar or necktie as if to loosen it. Occasionally, the sensation creeps up to the head, becoming, as one patient described it to me, "an expansion or swelling of the head—an opening and a shutting." Again, the epigastric sensation may be one of actual pain, which remains until consciousness is lost. Another very frequent symptom of the onset is that of a sinking or of actual pain in the præcordia, or violent palpitation of the heart. A feeling, identical with the *globus hystericus*, is also very frequently observed in epileptic insanity (*Gowers*). All these auræ, it will be noted, are referrible to a centric disturbance of the vagus and spinal accessory.

3. The Epileptic Paroxysm.—This paroxysm may be characterised by the predominance of the mental or motorial implication—that is, we may have transient, though complete, loss of consciousness, with little or no spasm; or the general convulsions may be the prominent feature, accompanied by early or later loss of consciousness; and since every shade of interblending of such phenomena may occur in different subjects, so no sharp line of demarcation can be drawn between the two extreme limits. A not infrequent symptom of the approaching attack is a non-painful contraction of the fingers of the arm first affected; thus one instance, amongst several, invariably has a painless flexion of the fourth and fifth fingers of the left hand with a cramp-like swelling of the right sterno-mastoid muscle which warns him that the fit is about to occur. These muscular cramps preceding epileptic seizures appear very frequent prodromata amongst the epileptic insane community. Classically, and for convenience of description we recognise the two forms, called respectively **le grand mal** and **le petit mal.**

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* *Loc. cit.*

(*a*) **Grand Mal.**—An aura may or may not precede, and the patient, if standing, may, without any warning, stagger to a seat, or fall suddenly down on the face or back, often seriously injuring himself. There may be the "epileptic cry," which is very frequent amongst the epileptic insane. It may consist in a subdued, plaintive wail, or a loud, wild scream, or a succession of piercing shrieks, as though the subject were actuated by terror; at times it is a mere hoarse gurgling in the throat, or a loud, prolonged groan; all probably due to the sudden forcible expulsion of air through a constricted glottis during the tonic spasms. The face is now deadly pale, the pupils dilate widely, and consciousness is completely lost. The convulsions beginning by tonic spasm, usually cause conjugate deviation of the head and eyes to one side, to which the body tends to roll; the spasm is usually more marked on one side of the body than the other; the chest is fixed, and respiration being arrested, the face becomes now injected and livid—the tongue, congested and swollen, is often forcibly protruded from between the teeth, the veins of the neck are swollen and rigid, and intense cyanosis prevails.*

The position of body and limbs will vary much in each individual case, depending upon the origin of the centric discharge, its strength and spread to collateral parts. Flexion and extension may be combined in different limbs—or flexion prevail throughout—the body being drawn up into a state of emprosthotonos. In the latter case the patient, if sitting or standing, almost invariably falls forwards; at other times the head is strongly drawn backwards, or backwards and to one side, so that the subject is twisted round in his chair as if looking over his shoulder. The tonic spasm now gives way very gradually to clonic convulsions—froth foams from the mouth, often tinged with blood, the tongue having been caught between the teeth and bitten by closure of the jaw; the fine vibratory character of the movement becomes coarser and broken up into rapid rhythmic movements, which eventually are large, interrupted, and cease entirely after a few irregular shock-like jerks of the limbs. The clonic spasms last from half a minute to two minutes, and after this cessation the patient lies stupefied and breathing stertorously. This, the third period of the fit, is very variable in duration, lasting from a few minutes to as many hours. There is a gradual return of normal breathing; sensibility and motor power are regained, and, with the exception of some heaviness and a dazed feeling, the previous condition of the patient may appear perfectly re-established.

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* Professor Bechterew has given the results of experiments suggested by him to Dr. Todorski, in which it is clearly proved that during epileptic seizures, artificially induced in animals, the tonic period is characterised by an increase of blood-pressure at both central and peripheral ends of the carotids, as well as in the jugular veins; whilst, in further experiments, it was shown that the pressure of the cerebro-spinal fluid always equalled that of the carotids. During the fit there is always an increased flow of arterial blood to the brain.—*Neurologisches Centralblatt*, No. 23, 1894.

The condition of the deep reflexes following upon these epileptic seizures is of interest. Certain seizures are invariably followed by exalted knee-jerk, and also well-marked ankle clonos; the clonos may be established a few minutes *after* the fit, or may succeed *immediately* to the convulsion. We have noted this *immediate* establishment of clonos also in cases of general paralysis, and, in one instance, in which severe epileptiform convulsions preceded by a cry affected the right arm only, excessive clonos existed in both ankles, but was most marked on the right side, the convulsive seizures being frequent, but of short duration, and followed by very prolonged unconsciousness. In contrast with the above there are other instances where the knee-jerk is completely lost after a severe fit, re-appearing again slowly in the course of four or five minutes; in such cases clonos is not observed, and the post-epileptic stupor has been prolonged and profound.

(*b*) **Petit Mal.**—In these attacks there may be nothing observed beyond momentary loss of consciousness and pallor of face. The patient may be sitting or standing during the attack; he does not fall. He may drop what he holds in his hands, or be suddenly arrested in movement, but may instantly recover himself, and act as if nothing unusual had occurred. Esquirol relates the case of a lady equestrian who had frequent attacks of petit mal when on horseback, yet never fell off. There was momentary arrest in her conversation, the bridle dropped from her hand, but, in a few seconds, she had recovered and finished the sentence interrupted by the attack. Very often the face, subsequent to pallor, becomes flushed (*Gowers*). In these slight seizures there may be slight facial spasm—the expression is momentarily *fixed;* or a spasm of the hand may occur, or a more noticeable (but limited) convulsion of very transient duration.

One of the premonitory *auræ*, before noted, with some vertigo and reeling may constitute such an attack. A patient may be subject to such attacks for years without a single seizure of *grand mal* occurring, or these two forms of epilepsy may occur indifferently, now one and now another, in the same subject—or attacks of *grand mal* interspersed with the minor attack, may gradually predominate and eventually wholly replace the *petit mal*. Thus one of our patients at the West Riding Asylum, subject to such seizures, was, while sitting up in bed one morning, requested to write his reply to a question; he wrote a lengthy answer, interrupted by some four or five such attacks. There was momentary loss of consciousness—the head drooped slightly, the pencil slipped through his fingers, but was almost instantly regained, and the sentence was continued without any apparent disconnection of words or displacement of letters; the interruption was so slight that, if he had not been closely watched, the condition might readily have been overlooked. In the case of this patient, a letter might have been easily written by him showing no confusion of ideas, and consistent in all respects, during a frequent repetition of such slight seizures as the above; and, in a medico-legal sense, this is of the utmost importance to recognise. At the same time, these slight attacks of epilepsy are well known to issue in the most rapid impairment of intellect—a fact recognised long since by Esquirol. This is because the disease is of the "very highest nervous arrangements in the whole nervous system, and of those which have the greatest integration, that is to say, of the substrata of consciousness" (*Hughlings-Jackson*).*

4. Post-paroxysmal Period.—It is during this post-paroxysmal period that much valuable information may be gleaned, as the mental automatism then displayed is in many cases prolonged, and affords us the opportunity of careful study. Epileptic mania of transient duration is a most common result of the paroxysm, but it is by no means always of so fleeting a nature. Cases occur where the maniacal excitement extends over many days without any further epileptic seizures intervening. It apparently bears no direct relationship to the severity of the attack, or to the number of epileptic seizures; it may follow slight seizures (*petit mal*), just as it may be the sequel to the major convulsive attacks (*grand mal*), and a *single* "fit," convulsive or non-convulsive, may leave the patient in this maniacal condition just as frequently as a *succession* of such attacks. This want of connection between the epileptic paroxysm and the occurrence of a maniacal outburst is perhaps more apparent than real. We are apt to lose sight of the fact that the slightest seizures are just the very cases where consciousness is prone to be most impaired or involved, and where a seizure is most likely to be wholly overlooked by the friends or even the patient himself; and thus it happens that a paralysis of the central hierarchy of the nervous system may so withdraw control over lower centres as to issue in wild excitement, although the epileptic seizure was so slight as to be scarcely, if at all, appreciable to the onlooker. In like manner the major discharge, *if it starts* (as in cases of insanity it most frequently does), from the highest cortical centres, may also leave these parts so paralysed as to result in a post-epileptic mania. One single attack may suffice for this issue; all depends upon the *site* of the disease being in the realms constituting the cerebral substrata of consciousness, and hence the vital importance of noting whether in suffers from epileptic excitement for a fortnight subsequently. She lies in bed in a huddled heap, covering her head with the bed-clothes, muttering incoherently. When disturbed, she utters an unintelligible jargon, interspersed with curses, and, showing her teeth with a fierce, vindictive look—half snarl, half grin—plunges beneath the bedding. In some cases, but *rarely*, the acute maniacal excitement sets in immediately upon the cessation of the comatose stage. In the case of a compositor, already alluded to, in whom garrulity augured an epileptic seizure, excitement has followed for a period of some eight or ten days. During this attack of epileptic mania he would wander aimlessly to and fro, or perform peculiar gyrations, talking incoherently in a loud, declamatory manner, and indulging in a rhyming propensity carried to a ridiculous extent.

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* *Loc. cit.*, p. 304.

Hysteroid Attacks.—These are not at all infrequent accompaniments of the epileptic seizure in the insane. Of these attacks Dr. Gowers says—"Instead of presenting such automatic action, some patients pass, as already mentioned, into a state of violent hysteroid convulsions. This sequel occurs chiefly at the age at which hysteria is met with, under thirty-five. It is most common in young women, frequent in boys and girls, occasional in young men. Hence it is evidently the result, not merely of the preceding epileptic fit, but also of the presence of the cerebral state which underlies the manifestations of hysteria."*

Case of E. C.

The most remarkable case we have met with occurred at the West Riding Asylum some fourteen years ago.

It was that of a young woman aged twenty-eight, and single, who continued for sixteen months under our observation, and who for the first twelve months was subject to epileptic and hysteroid seizures, whilst during the latter four months she was completely free from fits, and was discharged recovered. Her seizures occurred with great regularity every three weeks, and lasted from three to six days—the convulsive seizures occurring both night and day. The symptoms of the several stages were thus distributed.

Pre-paroxysmal Stage.—For some days before a seizure, restlessness, irritability, a pale and anxious look, depression amounting to despondency, and much insomnia, were noted. Then supervened a notable peculiarity of manner, and she confessed to aural hallucinations, viz.—a voice repeatedly calling out, "Kill them! kill them! kill them!" She was at this time distinctly suicidal and homicidal in her impulses, and always recognised that the "fits" were pending when the phenomenal voice occurred. Occasionally, without a seizure, she now became very violent and destructive.

Premonitory Period.—Restlessness more urgent, the "voice" more imperative, then the sensations of "a clock" within the head "wound up tighter and tighter," when all becomes dark, and consciousness is abolished. Asked afterwards to describe the "clock," she defined a circular sweep about 4 inches in diameter, and in a horizontal plane around the vortex of the head; and adds that if her hair were cut off she believes it would obviate the tight feeling of the winding. (*Her last automatic act after an actual seizure is that of pulling out her hair.*) Associated with the "voice," she occasionally hears bells ringing, and has a feeling of prickling pain within the eyeballs. She never refers to her suicidal impulses or to the "voice" which prompts her to the act until *after* the seizure; but, she often refers to the clock as it begins, crying out, "The clock," "the clock," and then becomes unconscious.*

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* *Op. cit.*, p. 692.

The paroxysm was invariably notable for the following features:—

(*a*) Deliberate rise of temperature and quickened pulse during the quarter of an hour preceding the fit.

(*b*) Peculiar recurrence of convulsive seizure in series of threes or sixes.

(*c*) Extraordinary periodicity of both diurnal and nocturnal seizures.

(*d*) Post-epileptic automatism replaced by hysteroid seizures occasionally; or by wild epileptic mania with determined suicidal or homicidal impulses.

The rise of temperature usually amounted to a degree above normal, occasionally higher; the pulse often rising to 130 before loss of consciousness; it was also peculiar in the fact that when the convulsion was arrested by chloral, *the rise in temperature still took place*, and became even more marked when the fit was thus suppressed. The attack of convulsions presented two varieties—in one, commencing almost simultaneously, and occurring bilaterally; in the other, beginning on the right side and spreading to the left, "Violent twitching of right angle of mouth; the head drawn slowly to the right: right arm affected by clonic spasm—then the left arm and hand; eyeballs drawn upwards; the feet raised from the floor, the left first, then the right, both rigid and quivering. Whole fit lasted about ten seconds, and terminated suddenly with complete muscular flaccidity." Another series of attacks is thus described. "Twitching of both eyes and angles of mouth; then clonic spasms of both arms and legs, a preliminary tonic spasm hardly observed; next, tonic spasm of the chest muscles with a loud scream; universal clonic spasms; relaxation of muscles, and fit over. Three or six such attacks occur in succession, the last always followed by closure of the eyes, which throughout the attack had been kept open." Then after three or more minutes' calm *the hysteroid attack* occurred; the eyes opened suddenly, staring vacantly, the head was drawn rigidly back, the body arched backwards in a position of opisthotonos for about fifteen to thirty seconds; then the head was thrown violently forwards and backwards several times, and a severe struggle ensued in which she had to be forcibly restrained by several nurses to prevent her rushing to the window or injuring herself or others. When watched (unknown to herself) in the padded room at night, the same attacks occur, and in the succeeding hysteroid seizure her body has been tossed from end to end of the room against the pads by the violence of the contortions; her bedclothes, also, after such attacks, were invariably torn to shreds.

The epileptic seizures above described maintained an extraordinary periodicity in their sequence; each series of fits almost invariably taking place at about the same hours throughout both day and night. Several features of the case would naturally suggest a purely hysterical origin for such an attack; but that the seizures were genuinely epileptic associated with sequent hysteroid seizures, was conclusively shown by associated conditions which were invariably present; these were—(1) initial rise of temperature and pulse; (2) aura of the "clock" followed by intense pallor of face; (3) extreme dilatation and inequality of pupils, the right always the larger; and (4) well-marked nystagmus.

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* This case, of which the main features are given here, has been published in detail in the *West Riding Asylum Reports*, vol. vi.

The number of epileptic attacks occurring in this subject varied from 80 to 120 (both day and night), and these became somewhat less frequent and less severe towards the fourth or fifth day as the termination of the attack approached. The following note made by the writer at the time illustrates the thermal disturbance preceding the attack:—"Immediately preceding the attack a rise of temperature occurred of 1° to 1·2° Fahr., and at the struggle a still further rise, often to the extent of 1°, or even more. The temperature then slowly fell again to 98·6°, and on one occasion as low as 98·2°, except when two fits succeeded each other quickly. On the occurrence of six struggles, the total elevation of temperature recorded was fully 2·2°, slowly falling to 99·2° during the following hour and a-half. *The previous administration of chloral invariably arrested the rise occurring before the fit; but at the exact moment when the fit was expected,* instead of a convulsive seizure, there was sudden profound sleep and a rise of about six-tenths of a degree."

Another peculiar phenomenon was noted after the convulsive seizures had ceased and whilst a still childish, pettish mood prevailed, with distinct alienation, the temperature taken at periods *corresponding to the time of her fits* was found from six-tenths to 1·8° above normal, although at other periods of the same day (not corresponding to the hour of a fit) the temperature was 98·4°. "As these periods arrived she would become greatly depressed, often starting up and complaining to the nurse of her low spirits." At these times (although naturally an intelligent, bright, and cheerful young woman) she would remain for days subsequent to the attack, childish in her pursuits, would make dolls' clothing, and fondle a doll like a child not out of her teens; was capricious in likes and dislikes, pettish and ill-humoured. This case illustrates in a forcible manner most of the very variable features of epilepsy. The premonitory depression; the tendency to distinct mental aberration; hallucinatory phenomena; the aura, epileptic, hysteroid and cataleptic states; impulsive homicidal and suicidal violence; and the resultant mental reductions following the more marked post-epileptic automatic stages.

Epileptic Katatonia.—We may here advert to cases of so-called **katatonia**—a term intended by Kahlbaum to include those multiple symptoms of stupor, cataleptiform, and ecstatic states, with phases of dumbness or reiterative speech—all running a certain cyclical course; and, according to this author, constituting in their *ensemble* a distinct morbid entity, as characteristic as general paralysis. Such symptoms are not infrequent in epileptic insanity; they especially prevail in the mental alienation of puberty and adolescence, in the puerperal forms of insanity, and are closely associated with the vice of onanism. The more closely we study these cases of katatonia described by Kahlbaum and other writers, the more convinced are we that we are dealing, not with any distinct pathological entity, but with some of the multiple phases of hysteria. Melancholia attonita closely approximates to the states to which we now allude.

Status Epilepticus.—The very extraordinary periodicity of the attacks narrated in the last case is, of course, exceptional; yet a well-marked tendency to the periodic return of epileptic seizures has long been recognised, and especially emphasised by Reynolds—"A large number of epileptics have their seizures every day, every two weeks, three weeks, and four weeks, while only a much smaller number suffer at such irregular intervals as cannot be thus expressed." This opinion can be endorsed by all who have had acquaintance with epileptic insanity, for it is undoubtedly true that these subjects exhibit a notable degree of such periodicity. A periodic recurrence is more frequent in the female than the male; but this is attributable to the associated menstrual derangements so often connected with the epileptic convulsion. The number of seizures varies greatly in some individuals; an enormous number have been recorded within short periods of time; thus a patient at the West Riding Asylum had 1,849 convulsive attacks recorded in a period of fifteen days; and Delasiauve mentions an epileptic who had 2,500 attacks in one month.* In general a patient has one, two, or three attacks during the day or night, recovering full consciousness between each seizure, possibly passing many days before a recurrence.

But if the attacks succeed each other rapidly, and consciousness be not restored between the convulsive seizures, if fit succeed fit at intervals of a few minutes only, the patient remaining comatose, we have developed what has been termed the **epileptic status**, a condition of most serious import. The pulse and breathing become quickened, and, as Bourneville first indicated, the temperature rises to 105° or 107°, with deepening coma and stertor; the patient is liable to sink. As the fatal termination approaches, the convulsions become more frequent, a few seconds only intervening between each discharge, so that at times they appear almost continuous, a fresh discharge being only recognised as a slight increase in the intensity of the convulsions. When this period arrives, however, the epileptic discharge becomes progressively feebler, and the fit may be characterised by a slight turning of the head and eyes to one side, with slight clonic movements of the limbs, or merely convulsive twitchings of one side of the mouth without conjugate deviation. The conjunctivæ are, of course, quite insensitive, the pupils being widely dilated and fixed to the strongest glare of light, while the face and body are bedewed with a cold sweat. Often the temperature exhibits unilateral deviations, being highest, by a degree or more, on the side first (or most) convulsed. If the patient recover, the fall of temperature is most rapid on this side until a balance is established; and subsequently, an equable and continuous decline of temperature proceeds on both sides. Many epileptics are subject to these occasional outbursts of convulsions passing into the *status*; these cases an observant medical officer soon learns to recognise, and experience teaches him the necessity of keeping them for prolonged periods upon bromide treatment. In the section on treatment of epilepsy we shall deal with this serious condition in detail; suffice it here to remark, that prompt and vigorous measures must at once be adopted if we wish to save our patient's life. The mortality from the *status epilepticus* is said to be due to (*a*) collapse, and (*b*) meningitis, the fits ceasing, the patient becoming delirious, developing bed-sores, &c. We cannot say that this latter termination has been seen by us; the mode of death has always been, according to our experience in asylum practice, exhaustion with hypostatic congestion of the lung.

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* Quoted by Ross, *op. cit.*, p. 932.

Epileptic automatism, of a most elaborate kind, is a prominent and often perilous feature in some epileptics after their fits; its interest as a medico-legal question is great. Thus we constantly observe patients at this stage perform not only the most incongruous acts, but carry out what would seem to be complicated purposive acts, to which they are entirely oblivious on return to normal consciousness. They will pick the pockets of fellow-patients; purloin articles in the most deliberate fashion; conceal weapons, such as knives, &c., in their pockets or beneath their clothing; and follow out, as before stated, a series of actions in acccordance with the promptings of some leading delusional idea, such as a somnambulist would perform. The case of *W. T.*, detailed below, illustrates this point forcibly, and still more so the case following it (*York Assizes*).

Interparoxysmal State.—We come now to the mental condition of epileptics in general at the periods intervening between their seizures, when the immediate effects of the attack are past, and prior to the disturbance engendered by the approach of a fresh series of fits. In fact, we have to study the peculiar characters of the epileptic neurosis, and the permanent mental reductions which become established, in consequence of the diseased state of the nervous centres and the disordered function. In reviewing a large number of the epileptic inmates of an asylum, it becomes evident that they may roughly be arranged in four classes.

(*a*) A small section is comprised by those who, upon the subsidence of the seizures, exhibit a perfectly normal state of mind; no emotional or intellectual disturbance can be traced by the strictest scrutiny, and their conduct (consistent in every respect) enables them to take up any employment for which they were fitted, and carry on responsible functions in various departments. Why are they, then, inmates of an asylum? Because their epileptic seizures are preceded or followed by such transient mental aberration, or by such reductions as render them *at these times* a risk to themselves and others; or, because the interval between their attacks is so short and exposes them to such risks in their usual avocations, that they demand continuous supervision and treatment. Outside an asylum this class is a large one; comprising, as it does, all those in whom the nature of the epileptic seizure is such as to affect the mental faculties but slightly, if at all, even in the pre-paroxysmal as well as post-paroxysmal stage. It is a well recognised fact, which the student must bear carefully in mind, that certain forms of epilepsy with frequent fits may last for many years, and yet the mental faculties remain, in the interval between the successive seizures, perfectly intact; nor must he be misled by any such notion (as we once heard expressed in a court of justice), that *because a man has had fits for many years* his mind must *necessarily have suffered permanently;* although, of course, in a large number of cases, the presumption is in favour of such implication.

(*b*) Then there are those cases of epilepsy in which the affective sphere of the mind is almost exclusively at fault; where, with a normal and often vigorous intellect, we still find, as a permanent residue, an emotional perversion, which maps them off from the healthy community, and which reveals itself by certain oddities, eccentricities of conduct and want of control; or, by an abnormal welling-up of feeling, an instability of emotions highly characteristic of the class. To this section, also, belong many who might be called moral imbeciles.

(*c*) Then there is the extensive class of those in whom the main feature is intellectual perversion; in whom delusional states are rife; and in whom the passions are violent and uncontrolled; a class which comprises some of the most dangerous elements amidst our asylum communities, since with all the natural impulsiveness of the epileptic, the delusional states engendered render them, at all times, apart from their paroxysmal seizures, prone to acts of desperate violence.

(*d*) Lastly, there are the advanced cases of epileptic dementia, in which the reductions are so extreme, that the higher emotions and moral sense are well-nigh extinct, and the intellectual operations correspondingly enfeebled; and in whom the mental life of the individual consists of the lower animal instincts and passions, and the impulses towards their immediate gratification. None of the insane arrive at a more degraded level than the epileptic dement; none of them exhibit more repulsive traits—more obnoxious passions; and in none does the physique undergo such a corresponding degradation in type.

Amongst the several arbitrary divisions thus enumerated, there are certain mental characteristics common to the whole class which largely enter into what we mean by the "epileptic neurosis." Notably prominent is the tendency to self-engrossment which may pertain, not only to the bodily sensations, giving rise to the grosser forms of **hypochondriasis**, but also to the passions, and feelings, and sentiments of the individual, which are morbidly dwelt upon and, so, intensified. Any bodily discomfort, however trivial, is thus apt to be exaggerated into a serious ailment, and incessant complaint is made to the medical attendant as to the state of the stomach, the bowels, the heart, &c. The epileptic is essentially a hypochondriac; on the other hand, irritability of temper, to which he is prone, is sure to find an object of complaint; imagined ills are conjured up, and he conceives himself the most injured individual in his ward. In like manner, his sentiments respecting his own abilities and aptitudes undergo a like intensification, and he becomes vain and self-laudatory. This rise in the self-consciousness begets an egoistic state of mind, which renders the epileptic the most selfish and narrow of all beings, and the corresponding decline of object-consciousness is well illustrated in his utter regardlessness of the time or comfort of others—his incessant and wearying importunity and demands upon the patience of his fellow-creatures, his obtrusive display of self-interested motives—in fact, in the profound decline of the altruistic sentiments and higher moral incentives to action. Opposed to this moral decadence, at first sight, might appear the statement that the epileptic often betrays a notable degree of religiosity; above all others of the insane, he is distinguished for his adherence to religious rites and formalities; importunate in his requests to attend religious services, addicted to repeating Scripture texts, to constant perusal of the Bible and devotional works, to singing sacred hymns, to falling on his knees in prayer upon inapt occasions and with an obtrusive show of mock piety; he but illustrates another phase of the rise of self-consciousness as it pertains to the religious sentiments. His religious life fails in its *intellectual grasp;* it is essentially egoistic, shallow, selfish, and similar to the undeveloped phases of the religious life in a low grade of civilisation. The grossest animal passions find their gratification *pari passu* with this mock display of pietistic fervour, with a sanctimonious bearing and a profuse indulgence in religious cant, and with apparent consistency in the epileptic's mind. The realisation of the religious life in action—the objectivising or actualisation which is its proper sphere—is at fault; there is a *decline in object-consciousness;* hence he finds no difficulty in reconciling these feelings with the continuous gratification of low and depraved instincts.

The lower types of epileptics also exhibit a characteristic low cunning and deceit; they are treacherous in their dealings with their associates, thievish in their propensities, and when arraigned upon a charge of misconduct, will meet it with the coolest audacity, and lie to the bitter end. The epileptic shows a tendency, akin to that of the hysteric subject, to malingering. Both will falsely accuse of violence those with whom they are aggrieved; will treasure up a tooth, or wilfully pull out their hair by the handful, and present it, to countenance their charge; and will cunningly call to their defence certain delusional notions to which they may be prone during the period of their seizure, if they can benefit their position thereby—this tendency should be carefully borne in mind. All the *apparent* delusional statements of an epileptic are not to be received, except with caution, as their sole object may be to obtain some indulgence or requirement, and especially so with the hypochondriacal subject. Consorting with this moral decadence the epileptic is eminently **instinctive and impulsive,** a feature demanding the utmost tact in his management at the hands of those who undertake his case; his conduct, when aroused, is peculiarly brutal and ferocious, and often characterised, like his actions during periods of **epileptic automatism,** by wholly disproportionate and **excessive violence.**

The **reaction-time** in epileptic insanity is delayed as will be apparent from the following series, taken indiscriminately from a large number of cases examined :—

Reaction-time in Epileptic Insanity.

	Acoustic Stimulus.	Optic Stimulus.
J. J. M., ...	·20 of a second.	·23 of a second.
J. V., ...	·21 „	·25 „
F. P., ...	·18 „	·23 „
J. D., ...	·19 „	·21 „
W. P., ...	·17 „	·19 „
R. H., ...	·24 „	·26 „
A. D., ...	·28 „	·29 „

Medico-legal Relationships.—No form of insanity so frequently presents itself to the medico-legal expert as epileptic insanity, and this from two very obvious reasons. Epilepsy is a disease to which the criminal class are peculiarly subject; it is the associate of intemperance, moral degradation, vicious bodily organisation, and the very varied heritage of a criminal parentage; and, in the second place, of all cerebral diseases it is the one which tends to engender impulsive forms of insanity, as well as to degrade and brutalise the victim's nature, whilst the phenomena of post-epileptic automatism often lead to acts of apparent criminality although the subject is really an irresponsible agent. First, then, we would ask : How far does the fact of epilepsy render its subject irresponsible for his actions? It is obvious from the foregoing considerations that epileptic insanity no more presents a *uniform* series of symptoms than do the physical accompaniments of the epileptic paroxysm always assume the same orderly sequence of events. Just as it is allowable to speak of epilepsies, rather than epilepsy, as regards the physical features presented by the attack; so the correlated mental symptoms exhibit very varied forms of insanity. And, apart from the varying type of the insanity, we also witness a great variation in *degree*: so that, we may not only find that our patient is prone to melancholia, mania, delusional insanity, impulsive insanity, dementia, but also that all these anomalies may vary in degree from the slightest to the most intense manifestations, or long periods may intervene wherein no mental anomaly presents itself. It cannot be questioned that many epileptics suffer little, if any, mental derangement prior. or subsequent, to their seizures; and, that the interparoxysmal period may be one consistent with the most perfect sanity, with vigorous mental activities, with intellectual capacities of a high order, and with special aptitudes and executive address which enable them to hold positions of trust and high responsibility. It is only as the *immediate forerunner* or *outcome* of the epileptic seizure that they may be truly irresponsible agents. The "fits" may even be of frequent occurrence, and yet the interval between two consecutive seizures may present no obvious mental derangement. We must not, therefore, assume that because a patient is epileptic and has many fits, even with mental disturbance, that he is necessarily alienated in the interval between such attacks, and therefore irresponsible for his actions. The longer the interval between two seizures, *cæteris paribus*, the greater the presumption also that the mental faculties may escape implication; and since frequent occurrence of fits is damaging to the mental constitution, especially fits of a certain type, so, conversely, we anticipate more interparoxysmal mental derangement in cases of rapidly-recurring attacks. In fact, the *proximity* of an act of outrage or violence to an epileptic seizure directly favours the presumption of mental impairment; and, in this connection, it must be strongly insisted upon that the mental disturbance following upon a single epileptic fit is frequently prolonged over many hours or even days.

The question might, therefore, be naturally put: if an act of violence be committed by an epileptic a day or two subsequent to an epileptic seizure, is the agent to be regarded as responsible for his conduct, because on the expiration of a further period he is found perfectly sane? Obviously, from what was implied above, we are *not* justified in assuming that, since he is free from obvious mental derangement a week or so subsequent to his seizure, he was not alienated for some hours, nay, days, after the attack. Acts of suicidal or homicidal nature may be committed subsequent to epileptic seizures as the outcome of

(*a*) Genuine automatism;

(*b*) Or as an incontrollable impulse devoid of motive;

(*c*) Or during the blind fury of epileptic mania;

(*d*) Or, lastly, the act may be instigated by the promptings of a deluded mind.

It is essential that we clearly distinguish these states in investigating the hidden springs of a murderous or suicidal attempt.

First, as regards **epileptic automatism,** it must be remembered that actions of very considerable complexity may be performed whilst the individual is a mere machine acting like a purely reflex mechanism, the patient upon return of normal consciousness being completely oblivious to the act which he has perpetrated; in this condition he is neither conscious of the act performed nor of its consequences.

Incontrollable impulse is another form of morbid activity which reveals itself in the subjects of epilepsy; like the motor explosiveness of the convulsive paroxysm, a **leading idea** may prompt to action with an imperative demand which brooks no denial. Epileptics are often conscious of this dire *necessity;* it may arouse within them the ancient doctrine of fatalism; they may be terrified at their own helplessness, and implore us to impose restraint—a plea the very last to be neglected by the medical adviser. The **impulsiveness** of the epileptic is proverbial, and should never be lost sight of in questions involving his responsibility; for, where other evidence of mental impairment is wanting, where delusion cannot be traced, where the subject was possessed of presumably normal consciousness at the time of his act of violence, still a factor of the gravest moment in this line of conduct may have been a notably diminished self-control. The essence of an impulsive act is, of course, its spasmodic suddenness and want of **apparent motive.** The lawyer naturally enquires for a motive, which, if found, he regards as evidence presumptive of the *volitional* nature of the act, and subversive of the doctrine of its *impulsive* character. He assumes that the presence of motive warrants him in regarding the epileptic as fully conscious of the deed he performs—of its nature and probable issue. We should be most guarded in accepting this conclusion.

The motives prompting to action in healthy mental operation are so complex as often utterly to defy our most careful scrutiny; much more so will this be the case when dealing, not with an organism which reacts within fairly constant or calculable limitations, but with the perturbed brain of the epileptic, in which the line of conduct is subject to no method of calculation. Even if there be a strong colouring of evidence that the act was the outcome of apparent motive, the natural, and often inborn, impulsiveness of the epileptic neurosis should warn us seriously against arriving at too hasty a conclusion upon this head.

In the third place, outrageous actions may be committed during the wild **mania** incident to epilepsy; in these cases, of course, no doubt can arise as to the agent's utter irresponsibility. The **nature of such acts** in these latter cases will often be characterised by their frightful violence; the crime can thus often be instantly identified by its blind, aimless, uncalculating, utterly reckless fury, which at once stamps it as the work of an epileptic (*Maudsley*). There are in these murderous outrages of epileptic mania indications of—(1) an utter loss of control, (2) of deep reductions in consciousness, (3) of violent explosive conduct.

Lastly, the act may be done, as stated above, at the **instigation of a deluded mind.** The epileptic insane are *not necessarily* (or even *frequently*) *deluded*, and we should look with some suspicion upon cases of affirmed delusion, fostered by those whose paroxysms are infrequent, or occur at long intervals. The delusions of epilepsy arise, as before stated, during the early and premonitory stages of the attack; the paroxysm itself often having the effect of clearing off the mental clouds, and of leaving the subject often better than before the seizure. One crucially important feature, however, to recognise from a medico-legal standpoint is, that the delusions prevailing prior to the epileptic seizure may be operative immediately subsequent to the fit, and before consciousness is completely regained. This has already been noted, but its importance merits emphasis here. When an epileptic suffers notably from delusion prior to his seizures, the outcome of his paroxysm should be carefully watched (*W.T.*).

It is, of course, of the greatest importance to recognise any connection existing between the conduct of the epileptic and the previously existing delusional state; since, if the act be the direct outcome of, or can be traced up to, such an aberrant state of mind, he must, of course, be regarded as an irresponsible agent. The depth of reduction in these epileptic derangements should receive attention.

(*a*) Was the act characterised by complete automatism?

(*b*) Or was he sufficiently conscious as to recognise its nature?

(*c*) Or was he sufficiently conscious to recognise its *criminal* nature also—the distinction between right and wrong, and the probable issue?

(*d*) Or, even if the latter was the case, was it the outcome of insane delusion, or perpetrated as a purely incontrollable impulse?

Malingering.—Epilepsy is, as is well known, frequently feigned by the criminal community; often with the object of exciting commiseration and extorting pecuniary assistance; and this is done with considerable cunning and success by some. But, though the community generally may be imposed upon with ease, it is scarcely possible that one well-versed in the subject could be deceived by the most cunning and expert. The intense pallor preceding the strong convulsions, the widely-dilated pupils, the disturbed organic functions, and, often, the minute extravasations of blood over the surface of the body cannot be assumed; and would, therefore, lead to speedy detection of the fraud. It is not so with the forms of mental derangement associated with epilepsy, the delusional perversions of this stage being readily counterfeited, and by no means easy of detection.

The plea of epilepsy is one so frequently established in defence of cases of outrage, assault, or murder, that the possible feigning of this disease and its forms of mental disturbance should always be borne in mind. The difficulty is greatly enhanced by the fact that the criminal classes are so much associated with those subject to epilepsy, that they acquire considerable address in feigning the disease; and they have sufficient cunning to assert the presence of hallucinatory and delusional states if thereby they can gain their ends.

And here we are face to face with another difficulty: the *genuine* epileptic is also notably cunning, and often much given to shamming —not bodily ailments alone, but mental also—usually with the object of obtaining some desired indulgences; it is by no means infrequent to discover an epileptic girl "shamming" a fit, just as others affirm they suffer excruciating pain, &c. Such a subject, arraigned on a trial of murder, would be most likely, if he thought the plea of insanity would save his life, to reproduce his former experiences, and assume delusions from which he might have suffered at times. In the case of *Reg.* v. *Taylor*, where the prisoner was charged with the murder of his infant child and of the police superintendent, it was believed that the statements advanced by the defence as evidence of delusional perversion (obtained *just prior* to his trial) were of this nature. The closest observation and repeated examination during his early imprisonment wholly failed to elicit a deluded state; and it is strongly suspected that the frequent subsequent examinations which he underwent suggested to his mind the policy of malingering. That he was fully aware of the gravity of his offence, and the probable issue, was made apparent by his statement to a fellow-prisoner on the night preceding the trial, that he would probably have to go to a lunatic asylum; a recognition of his position wholly inconsistent with the assumption of the defending counsel, that the prisoner was a complete mental wreck. That he was subject to delusions, about the period of his "fits," could not be doubted; and that the murderous act was instigated by such delusion is equally free from objection; yet the facts, that a period of some months had elapsed without such a seizure, and that no clue to delusion was forthcoming until just prior to his trial, were strong evidence in favour of his malingering. In this case also no epileptic seizure had occurred for three months subsequent to the murder; and the question as to the very existence of epilepsy in his case required examining. It was found that his neighbours and fellow-townsmen knew little or nothing about his "fits," and evidence as to such could only be obtained from interested parties—his wife, parents, and a lodger. But here again, on the other hand, it was obvious how readily a genuine description of epileptic seizures may be recognised from a feigned account. A most graphic account of *grand mal and* *petit mal* was given by each witness separately examined, consistent with each other in every detail, evidence which most distinctly would have broken down if the witnesses had not actually and individually witnessed the seizures. Another question of interest in this case was the actual condition of the prisoner's mind at the time of the act. Was the act characterised by impulsiveness, or was it the outcome of the delusions previously fostered? There is little room for doubt that the act was deliberate and intentional, according to his own account. He had for hours barred himself within his house, handling a loaded gun; his pockets contained several loaded cartridges; and it was only after watching his pursuers for some long time through the window of the house that he eventually took deliberate aim "behind the ear" of the police-superintendent and discharged his gun. He both intended to kill his victim and fully recognised the surrounding circumstances. In short, the act was very clearly *not* the *impulsive* act of epileptic furor, but the well-planned and determined act of a deranged mind prompted by delusion. One of the most striking instances of hallucination, or the aura epileptica, becoming the motive for action during the automatic stage is illustrated by a case where the subject (who was undoubtedly neurotic, of a very bad stock, but who was not known to have previously suffered from epilepsy), as he lay in bed beside his wife, imagined he saw two burglars rifling the contents of a chest in his room. He sprang out of bed, and, according to *his own statement*, as he rushed from the room for help, he saw one of the men rush upon his wife and strike at her with a hatchet. He remembers nothing more; but was found by a policeman (to whom he made the above statement) wandering in the streets, vacant and confused, and holding a hatchet in his hand. It appeared, from all the evidence produced in this case, that the poor man had a fit, preceded by the visual aura of the burglars in his room, that the idea of the hatchet prompted him to rush down stairs to the cellar in order to secure that weapon, and during this automatic stage he murdered his wife. No case could more forcibly indicate the frightful risk to which the aura may expose certain epileptics during the post-convulsive stage, and the necessity for close supervision.

Treatment.—No drug has so powerful an influence over the convulsive attacks of chronic epilepsy as the bromide of potassium, or the combinations of bromine with sodium and ammonium. The first-mentioned is most relied upon, and may be administered for very lengthened periods of many months without inducing *bromism*, and with very marked benefit. There are a certain proportion of the epileptic insane—doubtless the minority—in whom the bromides are of little or no avail; but by far the larger number exhibit a notable reduction in the frequency and severity of their fits upon their administration. The bromides have no immediate action in checking the fits, so that a somewhat prolonged treatment is necessary ere the desired effect is obtained; hence, if the attack is threatening (owing to the severity and rapid succession of the convulsions) to pass into the *status epilepticus*, it is of little use depending upon the bromide for cutting short the attack. For this purpose we have no rival to chloral, which, given in sufficiently large doses, rarely fails to arrest the seizures. Where there is an enfeebled heart and torpid circulation, large doses of chloral naturally suggest great risk—hypostasis certainly is to be feared; yet the imminent peril from exhaustion, due to the repeated seizures, renders it necessary to administer this drug, with certain precautions. It is well, first, to inject subcutaneously from $\frac{1}{120}$ to $\frac{1}{60}$ of a grain of atropine if a large dose (40 to 60 grs.) of chloral has to be given. Thus shielded, a sufficient dose of the drug may be given to completely arrest the attack, a procedure preferable, we think, to the more frequent administration of small doses. It is imperative in these cases that nourishment be given in the intervals between the fits; and, if the patient be too unconscious or torpid to swallow, it must be introduced by the stomach tube. In a few cases vomiting may occur and food so given be constantly rejected, and yet a nutrient enema may be retained, and, with this chloral may be combined. It should always be borne in mind that chloral has its *rôle* in the emergencies of epileptic outbursts, bromide in the more prolonged treatment. Many patients in asylums cannot live without the bromide treatment; if it be neglected, the fits become at once so frequent that they run imminent risk of passing into the epileptic status, and dying thus. Hence it is that in most asylums we find chronic epileptics who for years together, with short intervals of rest, are taking bromides continuously, who maintain their health well, have hearty appetites, are cheery and industrious, and whose fits, recurring at long intervals, would at once assume a serious character if the drug were suspended.

It has been stated that the percentage of hæmoglobin and the specific gravity of the blood occasionally increase after epileptic seizures, and are constantly higher in those epileptics who have for years adopted the bromide treatment, than in those who have taken bromides in moderation only.*

Prolonged treatment usually entails in many a very troublesome form of acne. It is customary, in such cases, to suspend the drug for a few weeks, and order the patient saline laxatives; but it may equally well be met by the combination of a small dose of the liquor arsenicalis with the bromide salt. In fact, arsenic may be given in all cases alike with decided benefit from the outset.

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* Vorster, *Op. cit.*

On the other hand, a certain proportion of our epileptic insane have a series of convulsive attacks periodically, often with intervals of months between. During the intervening period they are free from excitement, active, and cheerful subjects, but when once the fits are about to occur they become querulous, hypochondriacal, and violent. Such patients may often have their attacks cut short by a dose of chloral, and by removal from sources of irritation to the quiet of a darkened room; nor do they by any means invariably call for prolonged bromide treatment. To select those cases suitable for bromide treatment from those who can be safely kept without this drug, requires a prolonged experience—each case must be judged upon its individual merits; but, in all alike, bromide treatment should, in the first place, be adopted with the hope of possibly lessening the frequency and alleviating the severity of the attacks.

In the epileptiform attacks, such as characterise the history of many cases of general paralysis, we shall find that the bromides are of no avail; here chloral must be our sheet-anchor. The long-continued maniacal excitement of epileptics is best met by repeated doses of the asylum "green mixture"—*i.e.*, bromide in combination with the tincture of Indian hemp; half-drachm doses of the former, with one-drachm of the latter, given twice or thrice daily, rarely fail to alleviate the excitement. In the more serious delirious outburst of epileptic furor, it is well to administer chloral at intervals, followed by the former mixture.

How bromide acts upon the nervous centres we do not know; by what means it induces more stability of the discharging cells is at present a complete mystery. "Bromides are said to cause contraction of the small arteries of the brain, but it is exceedingly doubtful whether any part of their influence in epilepsy is due to this action" (*Gowers*).

Iron, given in combination with bromide, is of indubitable value in all such epileptics as exhibit any notable disturbance at the menstrual periods, at which time there is often not only a succession of fits, but also much maniacal excitement. Its use is also called for in all the hysteroid attacks.

GENERAL PARALYSIS OF THE INSANE.

Contents.—Prodromata—Egoism—Early Moral Perversion—Failure of Re-representative States—Enfeebled Attention—Transient Amnesia—Vaso-motor Derangements—Early Paresis—Second Stage—Delusions of the Paralytic and Monomaniac—Vanity and Decorative Propensities—Sexual Perversions—Facial Expression—Articulatory Impairment—Cerebral Seizures—Syncope—Epilepsy (J.F.)—Unilateral Twitching (J.S.)—Epileptiform Attacks—Conjugate Deviation—Case of H. P.—Apoplectiform Seizures—Monoplegiæ—Hemiplegiæ—Muscular Sense Discrimination—Apparatus for Testing Appreciation of Weight—Reaction Time—Spastic and Paralytic Myosis—Mydriasis and Amaurosis—Reflex and Associative Iridoplegia—Statistical Tables—Consensual Movements—Reflex Dilatation—Significance of certain Pupillary Anomalies—Spinal Symptoms—Deep Reflexes—Tabetic Gait—Incontinence and Retention—Atrophy of Vesical Muscle—The Blood in General Paralysis—Clinical Groupings of General Paralysis.

It is not an easy task for the student to gain a clear and comprehensive view of so protean a malady as that of general paralysis of the insane; nor need he be surprised or discouraged at this when he is informed that most authorities on the subject differ as to supposed varieties of the disease—whilst others are sceptical as to whether the term does not comprise *several* rather than *one* pathological entity.

General paralysis, even as a specific entity, has been called in question, and its severance from other forms of chronic cortical encephalitis has been regarded as an arbitrary and unjustifiable procedure. By far the most able attack upon the morbid unity of this disease has emanated from Dr. Reginald Farrar, and, although, we are not in accord with the general thesis, we fully appreciate the vigour of thought and breadth of view which characterise Dr. Farrar's article.*

When he is further told that no single portion of the entire cerebro-spinal system and its peripheral nerves (not even the sympathetic system itself) is safe from the encroachments of this far-reaching disease, he will be prepared to meet with a most complex symptomatology, and one in which varied groupings of symptoms may present themselves as one or other region of the nervous centres is implicated. Although the whole cerebro-spinal axis may become involved in this disease, it yet undoubtedly expends its chief force upon the cerebral cortex, which is primarily the affected site; yet, cerebral, bulbar, or spinal symptoms may one or the other preponderate, or be so variously grouped and associated, that several artificial subdivisions of general paralysis have been framed by different French writers of eminence, the utility of which, however, is questionable, except as a matter of pure convenience for purposes of description; they do *not* represent genuine pathological varieties. What the student should more especially bear in mind is the fact that, in this affection, he is dealing with a coarse brain disease, which, implicating primarily the highest nervous arrangements, is prone to spread progressively, both laterally and in depth; a disease which ultimately leads in all cases to dissolution of such nervous mechanism, and to correlated mental reductions. It must be admitted, however, that this progressive deterioration of the nervous mechanism from higher to lower, from lower to lowest levels, is regarded by some able observers as not a satisfactory statement of the case. Whilst readily admitting that the chief incidence of the morbid factor is on the cortical nervous system, some observers hold that the advanced, early, and extensive peripheral changes (nerve and muscle) so often found, and now universally admitted, bespeak something more than a mere *secondary* implication (secondary, *i.e.*, to the primary brain lesions) and that they closely approximate (if they are not identical) to the conditions found in the group of primary toxæmic Neuroses, dependent on an *intrinsic* toxic agent (*A. Campbell*).* The progressive impairment of highly elaborated motor mechanisms and the mental reductions comprise the characteristic features of this disease, however diversified in type. Moreover, his anatomico-physiological studies of the brain will have taught him that in a disease spreading over the sensory and motor areas of the cortex (involving so universally the substrata of the mental operations) the mode of onset, the signs and symptoms, the progress and duration, will vary greatly with the regions first implicated.

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* "On the Clinical and Pathological Relations of General Paralysis of the Insane," by Reginald Farrar, M.A., M.D., Oxon., *Journ. Mental Sc.*, 1895.

Prodromal Stage.—The prodromal stage of general paralysis is of very variable duration; it is usually prolonged over many months, and often embraces a period of several years. Many of the symptoms then apparent are trivial, taken by themselves; but several are of the gravest import and highly significant, especially when the *ensemble* is considered. A restless, unwonted activity (mental and physical) is of frequent occurrence, a feeling of superabundant energy prevails, for which there appears no adequate relief; often there is undue irritability and a perverseness which will not brook control or contradiction—an unreasonable demand upon the time and indulgence of others; waywardness, fickleness, or outbursts of furious passion upon trivial pretexts in those who had previously been more self-controlled and amiable; a growing change in the disposition and character, usually signalised by perversion of some one or more of the moral sentiments—a fact of primary import from a medico-legal point of view.

The implication of the affective sphere of mind may issue in melancholic gloom or despondency; or, on the other hand, in undue elation and *bien-être;* but just as often in sudden alternations of mood from one extreme to the other. The general restlessness spoken of pertains particularly to the ordinary pursuits of life and business; there is undue eagerness, a planning, scheming spirit, often exhibited in extravagant investments or in extraordinary outlay incommensurate with the subject's resources. Or it may show itself as intense anxiety about his prospects, his home and family. This frequently passes into more marked elation, an egoism which displays an exalted view of his own attainments in science, in art, or in general intellectual capacity ; an officious self-gratulation ; a tendency to extravagant talk, to laudation of his own status, his wife and family, and a yearning to test his intellectual or physical vigour. The religious sentiment is often in the ascendency, and may lead to various philanthropic schemes; and new projects may be based upon similar exaltation of the domestic or social feelings. Emotional waves are of frequent occurrence ; and silly, uncontrollable laughter may replace passionate weeping, for which no adequate cause can be assigned. It will be observed that we do not infer from all this a *distinctly deluded* state of mind—the existence of delusions becomes a more prominent feature in the subsequent stage—although at this period the patient hovers on the borderland of delusional perversion. The judgment is enfeebled and clouded (not necessarily perverted), and the condition is, in fact, one of overbalance. As before remarked, **moral perversion** is what appears so frequently to present itself at this period of incubation ; moral lapses are so frequent at this time that the unfortunate subject, especially if he belong to the lower strata of society, becomes lodged in prison and detained for offences committed during this early period of alienation. It is a most common experience in public asylums to receive from prison authorities subjects of this disease, who have been arrested for theft, drunkenness, violence, or indecent assault. The moral lapses to which we now refer differ essentially from the acts of those suffering from so-called moral insanity. In the latter, the actions indicate impulsive and uncontrollable states, as the result of a lowered or defective moral sense ; the normal inhibitory control is wanting and instinctive impulses rise into full activity. It is not so with the acts of the general paralytic ; they are neither premeditated nor impulsive, but casual, often appearing to be unconsciously performed ; even if the act appear determinate, its nature and consequences are wholly obscure to the agent's mind.

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* "Neuro-muscular Changes in General Paralysis," *Journ. Mental Sc.*, April, 1894.

And here the essential nature of these acts on the part of such subjects becomes apparent ; that high degree of **representativeness** essential for the recall of similar actions previously performed, and the vivid realisation of the consequences of such actions in the past, *is here wholly wanting ;* and still less is that **re-representative faculty** intact, which enables him to contrast the act as viewed in its nature with certain ethical canons. The moral lapse is, therefore, truly significant of a clouded intellect, of an **incipient dementia**—the cognitive, relational, or intellectual element of mind is on the wane. That such acts are not merely the result of simple perversions of the moral feeling is sufficiently attested to, by the complete absence of forethought and judgment which characterises them, by the absence of choice of circumstances favouring the act, by the want of object or reasonable motive—as when a wealthy man purloins an article of trivial value, as well as by the *silly* character of the act and manner of its accomplishment. An act of theft may be committed with open effrontery, no attempt at concealment being made; the most wanton outrage on public decency—the most audacious libertinism—may be committed by an individual apparently quite oblivious to a breach of public morals. Thus, a respectable member of society, of good social standing, gifted with many amiable virtues and natural talents, suddenly develops an unusual and objectionable freedom of speech and action, shocks his wife and family by various irregularities; plays the "hero to the barmaid;" indulges in unwonted alcoholic excesses; makes extravagant purchases or silly presents to *quondam* friends and casual acquaintances, for whom he suddenly professes a sincere attachment. In one such case, observed by the writer, extreme emotional instability prevailed, violent passion would ensue upon the most trivial occurrence, and just as readily might the patient be calmed into good humour, or made to shed tears profusely. Another patient, watched through this stage of the disease, conceived exalted notions respecting his family; his eldest daughter became a constant theme of converse, on which he would fondly dwell until he had utterly wearied his hearers. He then developed a too amiable weakness for the other sex, and from being a model husband, became careless, suddenly left his home, and was not heard of for some weeks. It then appeared he had developed a craze for preaching, and had travelled as an itinerant preacher amongst the mining community of South Wales. He returned to his friends deeply impressed with the importance of his mission; talked incessantly upon religious topics; and became morbidly depressed and hypochondriacal. In a case of incipient general paralysis, the subject of which was a highly talented mathematician, one of the earliest psychical symptoms was intense despondency, together with sudden lapse of attention and memory. Often when absorbed in the interest of solving a problem have we seen him cover his face with his hands; rise from his chair; and with a pained expression and the hurried remark, "It's of no use—it's all gone," hurriedly leave the room. He frequently confessed how painful such a state was to him; how utterly incapable he felt of exercising the slightest effort of attention; and how completely oblivious he became to the various links of the argument followed, before this disruption occurred. In this instance these sudden amnesic attacks prevailed for many months before definite aberration was recognised, and the onset of the established disease was one of sudden maniacal excitement, accompanied by acute hallucinations. This subject also spoke to his medical friends of the sudden and causeless emotional states—"as a welling-up of his feelings, only relieved by a passionate flood of tears."

The **transitory amnesic states** are very frequent as an early symptom of the disease, and almost invariably imply a serious failure in attention—the faculty which, as Sir J. Crichton-Browne has insisted, is *earlier* impaired than any other. To the same origin must be attributed the *forgetfulness* which is an invariable accompaniment of this early stage, and which so often leads to inconsistent, ludicrous conduct; inattention to the claims of others; and unconscious infringement of codes of honour, or of courtesy.

> "This loss of memory will be observable in many ways; especially is he likely to forget what he has done a day or two previously; and he will not only be forgetful, he will be careless, apathetic, and indifferent about that which formerly interested him; and, when he takes up new schemes and projects, his attention soon flags, and his interest vanishes. We see, in short, in his whole manner of life a weakening of mind, such as may be noticed in the commencement of senile dementia; but which, occurring in a fine and vigorous man of, it may be, thirty-five, too surely indicates the ruin even now commencing" (*Blandford*).*

We observe, at this period, that a very impressionable state of the vaso-motor system often prevails; palpitation with alternating flushing and pallor of the face, or, often, severe headache and neuralgic pains are complained of; the circulation is generally sluggish; and an early symptom (one for which the patient often first comes under notice) is that of a torpid liver. The hepatic functions are almost invariably deranged, leading to obstinate constipation, bulimia, and digestive troubles; the skin often assumes an icteric tinge; such symptoms affording material for hypochondriacal complaints; numbness of the hands, with tingling and formication of the skin, are also not infrequently complained of. Another frequent premonition is that of vertiginous attacks; slight attacks of vertigo often escape notice, the patient not complaining unless his attention is directed to the matter; they occasionally, however, become severe.

Even at this early date there may appear distinct motor troubles, a fine fibrillary quivering of the tongue may be observed, or a coarser twitching of individual fibres; an inco-ordinate jerky protrusion of the organ; a tremulousness of the upper lip or the facial muscles during conversation. Pupillary anomalies may co-exist also, or may antedate the above paretic symptoms by months or even years, as affirmed by Griesinger. Another highly significant group of symptoms is constituted by certain epileptiform or apoplectiform seizures which may now ensue, and which may become frequent at a later stage of the disease. They may usher in the fully-established affection, and thus may form, so to speak, a definite line of demarcation between the earlier and the second stage; but it is just as frequent to hear of such seizures, both convulsive and apoplectiform, far back in the history of the case.

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* *Op. cit.*, p. 260.

Second Stage.—After this stage of alienation has prevailed, for a longer or shorter period, more active symptoms are liable to arise; it may be by a *gradual transition;* but, often, there is an *abrupt* passage into a maniacal condition in which vivid hallucinations prevail. The **intensity** of the **excitement** is often extreme, acute maniacal states (verging even upon delirious mania) are frequent; incessant restlessness, obstinate sleeplessness, noisy boisterous excitement, and blind uncalculating violence, especially characterise such states. The reductions are so great that the subject wholly fails to appreciate the meaning of the simplest assistance rendered him; he struggles violently, and resists attempts to dress or undress him, or to give him the necessary food. His violence is often so great as to expose him to the most serious risk of fractured bones, even from the best-directed efforts to nurse and nourish him; such cases are a source of the greatest anxiety in our asylums. The blind fury of these states remind us of similar states of excitement in the epileptic; and, as a fact, are frequently a sequence of epileptiform seizures or of attacks simulating *petit mal.* In the less acute maniacal attacks the characteristic delusional state of mind reveals itself. With beaming face and muscles, tremulous from emotion, he endeavours to fix the glorious but transient visions which float before his mind's eye; in rambling incoherent utterances he insists upon his wealth, his exalted station or future destiny.

It will repay us to study a little more closely the nature of this expansive delirium. In the first place, the delusional state is the antithesis of the so-called *monomaniacal* delusion, which is essentially *fixed in character*, and is in itself a direct perversion of the individual's intellectual life. The grandiose conceptions of the general paralytic are wholly different in their nature, and are the direct outcome of an unrestrained imaginative faculty, no longer subject to the coercion of the reason. Those standards of objective reality which a life-long experience and knowledge may have established, no longer exist for him, or are clouded by the mental storm; and the only criteria of truth perceived are the subjective impressions aroused by the morbid excitation of his imaginative sphere of life; there is no reason why he should doubt their reality, as no challenge can be given by the overclouded reason, and so the sensuous procession of impressions pass by in everchanging kaleidoscopic hues, uniting and re-uniting in fantastic combinations, conjuring up visions of immortal life, of love, of beauty, of wealth, or of honour, or of all that mortal could desire. Challenge him upon the absurdity of his statements and a momentary irritation may occur; but he readily wanders off into his grandiose strain, asserting and re-asserting with stronger emphasis still more extravagant delusions.

It is in these states that the enfeeblement of attention is pre-eminently noticeable; faulty it was in the *earlier stage*, as we saw in the resulting mnemonic lapses and amnesic states; but, its failure now is a far more serious matter. The contrasting faculty of the mind, whereby a rational judgment can be formed, must decline with this enfeebled attention, since it depends for its existence upon the vigour of the latter. This failure of attention can be occasionally elicited in a remarkable manner as regards certain special mental operations. It is readily observed upon testing this faculty that it occasionally fails more with certain mental operations than with others—naturally with those less habitual to the subject—and, if we continue to test the patient in this direction, the strain becomes at times intolerable, and has a strange result. Thus in a patient, who was garrulous and optimistic, talking incessantly upon the subject of his "coursers and blood-horses," it was detected that he could not direct his attention to simple numerical calculations without much painful effort; upon one occasion, therefore, when his attention was forcibly directed towards a simple sum of addition, after giving a wrong answer once or twice, the effort resulted in a sudden (but transient) loss of consciousness, a twitching of the facial muscles and right hand, and an aphasic state lasting some five minutes after regaining consciousness. The following day a similar test was applied to this patient *with identical results*, except that the convulsive discharge was spread over a wider range.

We have recorded a somewhat similar case where the patient, who was suffering from progressive paralysis, could not protrude his tongue without inducing violent left facial spasms, the tonic twitchings being associated with much vaso-motor paresis.*

Beyond the fact that the delusions of the general paralytic are so *transient* and *variable*, there is their *simple*, *sensuous*, *and fragmentary nature* to be noted; they bear no logical connection the one to the other, and are therefore most incongruous and self-contradictory. Then, again, such delusions are *simple assertions*, the general paralytic does not *reason* out his delusive concepts, or attempt to erect a system of belief thereupon; he simply asserts, re-asserts, and never attempts a proof. Herein again we see the distinction between his delusions and those of the monomaniac. In normal states the imaginative faculty, however active, if duly controlled by reason, may find its expression in poetic imagery or on the painter's canvas; but in the case of general paralysis, emancipated from such guidance, its vagaries become so astounding that they defy expression. We all know how the very indefiniteness of emotional states renders their expression by language difficult, and at times impossible; and how, in contrast with the feelings, the subjects of exact knowledge find a ready medium for their expression and elucidation in the faculty of speech. The mental life of the general paralytic at this stage is so far made up of sensuous feelings and their residual emotions, that he wholly fails to his own satisfaction, to express by language what rises before his mind, his feeling and mental imagery are illimitable, and submit not to the definition of words. Thus, in each repeated utterance, he tries to rival his former extravagance; he has not simply millions, but "thousands of millions of millions of millions."

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* "Ocular Symptoms of General Paralysis," *Brit. Med. Journ.*, April and May, 1896.

The nature of our patient's occupation, and the subjects whch have chiefly engrossed his mind, will usually afford material for these delirious conceptions; thus, a poor labourer who through years of anxious toil has struggled to support a large family believes that he has accumulated enormous wealth—"is heir to extensive domains, and his children princes of royal blood;" another, a schoolmaster, talks on schemes of universal education. One who had squandered his means upon the turf was the imagined possessor of twenty hunters which he had just sold for £350 each; another, a poor carter, is possessed of a magnificent team of horses, each of which he calls by name, and excitedly smacks an imaginary whip, as he drives them on in mad career. One who had occupied a foreign diplomatic post had conceived extraordinary schemes for developing the industrial and mercantile resources of all the European nationalities. Some are agitated by vast philanthropic schemes; one of our patients was going to empty all the prisons, asylums, and workhouses in England, and start each individual afresh in life "upon a sovereign each;" another intended paying off the National debt. The exuberant welling-up of feeling transforms the status and surroundings of the subject without affecting his real identity; he still retains his name, but is now a duke, a king, or emperor; his wife and children still are his, but are exalted into corresponding dignities; whilst the asylum is a gorgeous palace, the nurses or attendants transformed into princes or courtiers.

Fantastic decoration is much indulged in, especially by the female paralytic; scraps of coloured stuffs, ribbons, and coloured paper are stitched on to their clothing as insignia of distinction, or as an addition to the attractiveness of the subject. The sexual characteristics are prominently developed; the female, especially, betraying much personal vanity or much self-consciousness in the presence of the opposite sex; she is often engaged on matrimonial alliance; connubial subjects occupy the chief theme of her delusions; and, occasionally, a well-marked erotic state prevails. On the other hand, the male paralytic raves upon wealth, property, social position, professional attainments, manual dexterity, artistic ability, muscular power, and endurance. The variety and transient nature of these delusions; their utter silliness, impossibility, and inconsistencies, indicate a serious degree of dementia in which an enfeebled attention and an unbounded license of the imaginative faculties coexist. The associated excitement may at times be in abeyance, to be called up readily upon the slightest reference to optimistic or grandiose subjects, when the stolid, half-vacant expression lightens up into a look of fatuous rapture as he pours out his delirious notions. Even in the calmest moments an undercurrent of excitement usually exists, especially manifested in restless, purposeless movements and misdirected energy, with nocturnal exacerbations, during which he is noisy, destructive of clothing and bedding, and dirty in his habits.

It is at this stage of our enquiry that two groups of physical signs become prominent features; present as they may be in the earliest stage of the disease, they are, however, almost invariably found at this period. They consist in certain articulatory troubles and oculomotor paralysis. Grandiose delusions with maniacal outbursts, a **delire ambitieux,** are by no means an unusual feature in some other forms of insanity; but when to this delirium there is superadded a tremulousness of the lips, an inco-ordinate ataxic state of the tongue, and certain pupillary anomalies, the diagnosis of general paralysis is next to conclusive. The **facial expression** of the general paralytic is characteristic; when unmoved by emotional excitation there is great stolidity, with a somewhat vacant demented aspect, but when roused into conversation the face beams with emotional excitement, the lips and facial muscles become tremulous, and twitchings of the muscles of the brow are noticeable. The tremulousness of the lips is well compared by Dr. Bucknill to the like tremulousness in a person about to burst into a flood of tears. An uneasy fixity of the lips is noticed in some, and a tendency to place the hand over the mouth whilst speaking, in full consciousness of the failure. When the tongue is protruded, it is with ataxic jerks or irregular inco-ordinate movements; and a fine fibrillar tremor will be perceptible whilst it is extended. In *advanced* cases the tongue will be protruded only with great effort, the mouth being widely opened, the eyes staring, and the whole head trembling and unsteady with the effort expended. The **articulation** is now distinctly impaired.

Articulatory Troubles.—The character of the articulation is distinctive; it is slowed, hesitating, blurred, approaching that of a drunken man; its utterance is broken, syllabic recurrences are interpolated, and the difficult enunciation may end in an explosive effort. In the early stages of the disease, however, a slow, laboured enunciation, with slight blurring of consonantal sounds, may be all that is recognisable; but, upon excited converse, as when rallied on the subject of his delusions, the impairment may be at once exaggerated and accompanied by the characteristic tremor of the upper lip. It is the labial and lingual utterance which suffers chiefly; and, if present, it is at once manifested by requesting him to repeat any alliterative doggerel; to repeat distinctly such words as hippopotamus or perambulator. The paralysis of lips and tongue advances in later stages to a more profound degree, and attempts at speech issue in an inarticulate muttering of broken unintelligible jargon, in which here and there some word is recognised.[*]

"Cerebral Seizures."—During this stage, or later still, the patient may be subject to convulsive, apoplectic, or paralytic seizures; and very few indeed pass through the descending series of dissolutions of general paralysis without suffering from one or more of these accompaniments. Such "seizures" are—

Syncopal or quasi-syncopal attacks.
Petit mal, or, exceptionally, *grand mal*.
Limited (or unilateral) twitching.
Epileptiform discharges.
Apoplectiform (or true congestive) attacks.
Hemiplegiæ and monoplegiæ.

Syncopal Attacks.—These are by no means infrequent during the progress of general paralysis, and are often the first warnings given of a failing heart, and of the necessity for keeping the patient in bed. Thus a patient taking his customary meal will suddenly turn pale and fall forwards; his pupils are dilated, his pulse imperceptible, and the skin cold and damp; no convulsive twitching occurs, and after a momentary prostration, he rallies and recovers his former state. Such patients demand rest in bed. Attacks of *petit mal* are occasionally mistaken for syncopal attacks, and reported as slight "faints" by the nurse or friends.[†]

Epileptic Seizures.—These are of frequent occurrence, yet by no means so frequent as the epileptiform and limited convulsive attacks. They also occur in early stages of the disease, and are usually referred to by the friends of the patient as slight faints. Attacks of the nature of *petit mal* are the more usual. They are characterised by very transient loss of consciousness preceded by pallor, wide dilatation of pupils, and perhaps a slight twitching of one side of the mouth, followed by much confusion of thought, obvious in inconsistent speech and conduct; or by more prolonged automatic states.

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[*] See "Hemiatrophy of Tongue, with Numerous Cases," by Trevelyan, *Brain*, vol. xiii., p. 102.

[†] See "Warnings of General Paralysis of the Insane," by George H. Savage, M.D., *Brit. Med. Journ.*, April 5, 1890.

J. F., March 21, 1881, seized this morning with convulsions, which occur every ten or fifteen minutes, and are identical with epileptic seizures, except that the convulsions are chiefly unilateral, involve the chest muscles but slightly, there being also no lividity of face nor obstructed breathing; each attack lasts for thirty seconds or thereabouts.

The convulsive phenomena in their sequent stages were as follows:—

1. No pallor, but head and eyes deviate to the right; there is a broken inarticulate cry; the pupils dilate widely; the brow is raised by the occipito-frontalis.

2. The mouth is drawn to the right, lips twitch strongly and uncover the canines.

3. Right arm flexed, with forefinger extended; then raised and convulsively jerked at shoulder; the brow twitches violently.

4. In certain seizures the discharge spreads to the right leg also, but did not involve the left.

After the fit there was paralytic deviation of head and eyes to the left and notable helplessness of the right arm; the left pupil was much larger than the right, but slowly regained its former size; there were champing movements of the jaw; no exaggeration of patella-reflex; no ankle-clonos; at the onset of each attack the heart, previously beating strongly, became imperceptible during the tonic stage. The cry always precedes each attack.

On the other hand, the occurrence of general paralysis as a sequence to ordinary epileptic insanity is very rare, a fact which has also been indicated by Savage, who states:—"It is remarkable that the epileptic insane provide very few cases of general paralysis, but that general paralysis follows epileptiform fits in many cases. So much is this my experience, that when I hear of the onset of fits of the epileptic type in a middle-aged man, I at once look for other signs of degeneration."*

Limited or Unilateral Twitching.—Sudden, rhythmic twitching of the muscles about the mouth, or of the specialised groups of the hand, or of the forefinger and thumb of one side are very frequent, either alone or in combination; or convulsive twitching of the flexors of the wrist or elbow may also be associated therewith. The various muscles of the thigh or leg may be observed picked out by the convulsive discharge, or the whole arm or leg jerked spasmodically. Such twitching is often increased by handling and passive movement of the limb. The limb may be fixed in rigid extension, whilst the toes or fingers are flexed by clonic movements. The muscular twitching may be very general, involving both sides of the body (although unequally); and its duration may be protracted over days or even weeks without interruption. The following case illustrates this fact:—

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* *Op. cit.*

J. S., a general paralytic, was seized with convulsive twitchings of the limbs on the 25th of November, 1886. His face was flushed and the skin covered with a greasy unctuous sweat. Both arms, but especially the left are continually and consentaneously jerked by the convulsive twitching of the extensor group for the elbow and wrist, the fingers of the left hand are suddenly spread as in the act of playing the pianoforte; the toes also show a tendency to "spread," the feet being rigidly extended, whilst there is almost continuous clonos of both ankles, especially increased by flexion of the foot; if, during a period of partial cessation of this clonic state, the sole be irritated by a pin, clonos is again briskly established. There is a notable degree of the "paradoxical contraction." The superficial abdominal reflexes are dulled. *Tache cerebrale* is rapidly produced, and is vivid over all parts of the body. Both conjunctivæ are injected; both pupils show mydriasis, but the left is larger, and both are fixed to a bright beam of light. Bowels and bladder paralysed. Patient is greatly demented and quite mute.

The following day the twitching was limited to the left foot and hand; the same expansive movements of the digits occurring.

November 30, 1886.—The convulsive twitching of the left hand and foot remains unchanged; the plantar reflex is greatly exaggerated and hyper-sensitive.

April 16, 1887.—The movements above described have continued up to this date (nearly five months) without interruption, but are now gradually declining.

The above was, of course, an instance of such convulsive movements in an advanced stage; but similar seizures may occur at a very early period of the disease. The convulsive twitching is usually associated with a certain degree of reduction in consciousness; and, when the discharge involves the right side of the face and hand, **aphasic states** may prevail, and a certain degree of **word-blindness** or **deafness** presents itself.

Epileptiform Seizures.—Under this term are comprised general convulsive seizures, or convulsive discharges from motor centres, representing large associated groups of the musculature of the body and limbs. Such attacks are often ushered in by premonitory twitchings, such as those just described; they are not, as a rule, accompanied by complete loss of consciousness. The convulsion or spasm may start, as indicated by Mickle, "from some point as it were, becoming widely spread and severe, then ebbs away and ceases everywhere except at the starting point, usually the mouth, eye, or hand, where occasional jerks are seen which may gradually die out; or, on the contrary, the preceding cycle of events may be repeated, or the renewed convulsion may chiefly affect the other side." *

The onset of the attack is almost invariably hemispheric—*i.e.*, the convulsions begin unilaterally, and may or may not spread to the opposite side; they are often preceded by a well-marked tonic stage as the rapid primary discharges occur; the clonic stage being often long protracted, becoming more and more broken-up into intervals of comparative rest until at last an occasional convulsive jerk of the limb or separate muscular contractions alone prevail.

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* *Treatise on General Paralysis*, p. 163.

Another form occasionally met with is that of an *associated movement* of the head and arm; the head and eyes turn as if looking over the shoulder, the pupils dilate widely, and the arm of this side is simultaneously raised in the same direction with the forefinger extended, and a painful cry escapes the patient. This movement may be repeated over and over again for hours in succession. It is important to recognise this form as occasional in general paralysis since it has been affirmed that the *cry* is a distinctive feature, thus:—"Patients in the fits of general paralysis seldom bite the tongue, the convulsions are not so violent, there is not the aura, *nor the cry*, and the mental symptoms will of course be quite different."* (*Blandford*).

These convulsive attacks will be followed by the usual results observed after severe discharges from cortical grey matter. There will be partial, or more or less complete, paralysis of the muscles involved; the facial muscles may be involved, the cheek flattened, and the mouth drawn to the opposite side; there may be more or less **glossoplegia**, or the patient may be completely aphasic with right **brachial monoplegia**; or the leg only may be temporarily paralysed, or hemiplegia may prevail. **Conjugate deviation** of the head and eyes is also frequent as a post-convulsive sign.

Epileptiform seizures may occur at an early period of the disease, usually not until twelve months have elapsed. According to Dr. Newcombe, out of 100 general paralytics, 51 patients suffered from epileptiform seizures, and of these 51, as many as 19 did not develop such symptoms until between twelve to twenty-four months after the commencement of the disease, whilst one only had convulsions within three months of the onset.†

The immediate result of these general, wide-spread, epileptiform convulsions is of far greater import as affecting the mental aspect of our patient. They usher in the gravest reductions, often leaving the subject a complete mental wreck. Take for instance the case of

H. P., who was in the second stage of general paralysis, was mildly excited, and the subject of extravagant, grandiose notions, yet retaining a fair amount of mental energy sufficient to enable him to read, write, or to converse in a connected strain of thought, so long as his delusional ideas were not entrenched upon. He was suddenly seized with epileptiform convulsions, commencing on the left side of the body, but usually spreading to the opposite side; such seizures occurring several times in the course of the day and night, and lasting for several days together. On their cessation he was left in a condition of profound imbecility, from which he never rallied. In his case, persistent and copious watery alvine evacuations accompanied the convulsive attacks.

The mental deterioration following epileptiform seizures is often so notable as to sharply demarcate the stage of maniacal excitement and delusional perversion from the last stage of hopeless dementia and motor helplessness.

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* *Op. cit.*, p. 266 (Italics not in original).

† See "Epileptiform seizures in general paralysis of the insane," *West Riding Asylum Reports*, vol. v.

The epileptiform seizure is also ominous of rapid break-down; "in twenty-four out of sixty cases, death occurred within a month after an attack" (*Newcombe*).*

Apoplectiform Seizures.—The patient may be struck down suddenly by symptoms of an apoplectic type, associated occasionally, but by no means necessarily, with slight convulsive discharge. He becomes helpless in his limbs, heavy, lethargic, and stupid, and this state may deepen into complete coma. The face is deeply flushed, the head hot, and the body generally bedewed with perspiration; the breathing may be heavy and laboured, the pulse rapid, and the temperature quickly rises to 103° or higher. The condition is always a critical one, there being hypostatic engorgement of the lungs and pneumonia threatening the patient's life; if this is not fatal, it is always followed by serious results—viz., by various motor paralyses, the advance of **dysphagic** symptoms, and by much increased mental enfeeblement.

Monoplegiæ and Hemiplegiæ.—Paralysis suddenly occurring without apoplectic or epileptic premonitions involving one or both limbs, or complete hemiplegia, of *very transient duration*, is another feature frequently occurring in the course of this disease. The *suddenness* of onset is notable; the hand drops whilst at work utterly helpless; or the patient suddenly stumbles whilst walking, and is found paralysed in one leg; the deep reflexes will be exaggerated and ankle-clonos prevail. Patients will thus be found after a quiet night's rest suffering from a **crural** or a **brachial monoplegia** which may have completely disappeared in a few hours or days.

Muscular Sense Discrimination.—The localisation of the sense of muscular discrimination in the so-called "motor area" of the cerebral cortex—the "kinæsthetic centres" of Bastian—appears lately to have received considerable confirmation from the clinical and the pathological side. We shall not attempt here to do more than to indicate that in general paralysis where the kinæsthetic centres are early, and in the majority of cases most seriously, involved, the muscular sense is obviously defective, and the delicate appreciation by this channel often very seriously blunted. We are not aware that this question has been definitely settled prior to this date, and we, therefore, bring forward our results as suggesting (1) the localisation of this sense, and (2) the means of accurately determining the defect in this sense by measurement.

In the following cases of progressive paralysis of the insane, the muscular discrimination was tested in right and left hands, with the most careful precautions to exclude all fallacies, and all instances were omitted where dementia was so advanced as to introduce a chance of error. The instrument used was Galton's, a description of which is given on p. 301. The figures in the tables indicate the respective indices of the finest series successfully arranged : thus 6—6 indicates that series 6, and all the coarser series up to 12, were successfully dealt with, but that at series 5 the operator failed, and, therefore, series 6 represents the measurement of his bluntness of muscular sense appreciation.

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* *Loc. cit.*

MEASUREMENTS OF MUSCULAR SENSE APPRECIATION IN G.P. LOWEST REGISTRY.

	Right.	Left.		Right.	Left.
E. C., ...	3	7	J. S., ...	8	8
C. A. (alcoholic excess), .	3	5	J. V. B., ...	8	8
J. C. (alcoholic excess), .	3	4	J. H^2., ...	8	10
W. G. H., ...	4	12	W. G., ...	8	9
T. B., ...	5	5	E. B. (alcoholic excess), .	8	10
J. H., ...	5	6	E. A. A., ...	8	10?
S. H., ...	5	8	S. H^4., ...	8	8
J. W. H. (extremely grandiose),	5	8	E. B. (alcoholic excess), .	8	8
S. H^2. (alcoholic excess), .	5	5	T. V., ...	9	9
W. R., ...	5	7	W. B^3., ...	9	9
E. S., ...	6	6	C. S. R., ...	9	9
T. C., ...	6	8	T. S. (alcoholic excess), .	9	12
C. W. (alcoholic excess; much tremor of hands), . .	6	8			
R. D., ...	6	7	R. B. (alcoholic excess; syphilis; manual and locomotor ataxia), . . .	12	12
W. B., ...	6	6	J. M. (alcoholic excess), .	12	12
W. H., ...	7	7	J. H^3., ...	12	8
W. B^2., ...	7	6	T. S^2., ...	*fails at* 12	*fails at* 12
J. E. (much hypochondriasis),	7	7	W. R. (syphilis), ,,	12	*correct* 10
S. H^3., ...	7	9	T. M., . . . ,,	12	,, 9
T. L. N., ...	8	9	W. G., . . . ,,	12	*fails at* 12

How far this failure of the appreciation of weight could be dissevered from loss of tactual and other sense anomalies, was put to the test in several cases, with the results indicated in the following table. In most of these we perceive that little or no impairment of cutaneous sensibility existed, but, on the other hand, an acute tactual sense, and a fine discrimination for temperature and painful impressions. The discrimination between slight differences in temperature was tested by tubes held in the palm of the hand, into which cold or warm water was gradually poured to obtain the delicate shades of warmth required. Tactile sensibility was tested by the ordinary æsthesiometer; the results with the latter instrument sufficiently attest to the absence of dementia as an element explanatory of the muscular sense measurements. For comparison with the table of measurements of the tactual sense we give the *normal results* as stated by the best authorities.

(*a*) Tip of forefinger,	2·0 mm. to 2·3 mm.	(*d*) Back of forearm, . .	45·1 mm.
(*b*) Ball of thumb,	6·5 ,, to 7·0 ,,	(*e*) Front of forearm, . .	15·0 ,,
(*c*) Back of hand,	31·6 ,,		

Cases of General Paralysis.	Muscular Sense Discrimination.		Tactual Discrimination.*		Sense of Temperature.	Remarks.
	Right.	Left.	Right.	Left.	Right. Left.	
W. B^2.,	7	6	(*a*) 2.5 mm. (*b*) 12·5 ,, (*c*) 7·5 ,, (*d*) 22·5 ,, (*e*) 20 ,,	5 mm. 7·5 ,, 10 ,, 12·5 ,, 12·5 ,,	Exquisitely keen.	Excited and very grandiose.
J. E.,	7	7	(*a*) 5 ,, (*b*) 9 ,, (*c*) 12·5 ,, (*d*) 20 ,, (*e*) 22·5 ,,	5 ,, 9 ,, 10 ,, 17·5 ,, 22·5 ,,	Do.	Depressed and hypochondriacal.
S. H^3.,	7	9	(*a*) 2·5 ,, (*b*) 3·5 ,, (*c*) 6·5 ,, (*d*) 11·5 ,, (*e*) 3·5 ,,	2·5 ,, 5 ,, 9 ,, 22·5 ,, 5 ,,	Do.	Advanced G. P. with extreme tremors of face and limbs.
E. B.,	8	10	(*a*) 2·5 ,, (*b*) 7·5 ,, (*c*) 10 ,, (*d*) 15 ,, (*e*) 15 ,,	2·5 ,, 10 ,, 7·5 ,, 17·5 ,, 17·5 ,,	Do.	G. P.—Alcoholic excess.
S. H^2.,	5	5	(*a*) 3·5 ,, (*b*) 5 ,, (*c*) 10 ,, (*d*) 12·5 ,, (*e*) 15 ,,	2·5 ,, 5 ,, 5 ,, 17·5 ,, 22·5 ,,	Do.	G. P.—Alcoholic excess.
J. H.,	12	8	(*a*) 2·5 ,, (*b*) 5 ,, (*c*) 7·5 ,, (*d*) 10 ,, (*e*) 10 ,,	1·5 ,, 2·5 ,, 10 ,, 7 ,, 12·5 ,,	Do.	G. P., with extreme hypochondriasis.
W.G.H.,	4	12	(*a*) 2·5 ,, (*b*) 10 ,, (*c*) 12·5 ,, (*d*) 12·5 ,,	2·5 ,, 5 ,, 12·5 ,, 7·5 ,,	Do.	
C. S. R.,	9	9	(*a*) 2·5 ,, (*b*) 10 ,, (*c*) 15 ,, (*d*) 12·5 ,,	2·5 ,, 10 ,, 10 ,, 10 ,,	Do.	
R. D.,	6	7	(*a*) 2·5 ,, (*b*) 10 ,, (*c*) 15 ,, (*d*) 12·5 ,,	2·5 ,, 7·5 ,, 12·5 ,, 17·5 ,,	Do.	
C. A.,	3	5	(*a*) 2·5 ,, (*b*) 10 ,, (*c*) 15 ,, (*d*) 15 ,,	2·5 ,, 7·5 ,, 15 ,, 12·5 ,,	Do.	G. P., with history of alcoholic excess.
T. M.,	fails 12	succeeds with 9	(*a*) 2·5 ,, (*b*) 6·5 ,, (*c*) 7·5 ,,	2·5 ,, 9 ,, 7·5 ,,	Do.	Extreme tremor of both hands.

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* The above figures for tactual discrimination express in *Millimetres* the minimum distance at which two points of the compass are distinguishable.

These results clearly imply for all the cases of general paralysis examined, a keen sense of slight differences of temperature; but slight or no impairment of tactile sensibility, with indeed much exaltation of the latter at certain sites—*e.g.*, at the back of the hand; and, on the other hand, distinct impairment of muscular discrimination.

It is, therefore, interesting to note that in certain other cases of insanity a similar failure prevails, and where we have every reason to believe similar regions of the cortex are involved. We refer to cases of adolescent insanity complicated with masturbatic habits, and to certain cases of alcoholism and epilepsy. The following measurements may suffice to illustrate these facts:—

MUSCULAR SENSE IMPAIRMENT IN ADOLESCENT, ALCOHOLIC, AND EPILEPTIC INSANITY.

	Muscular Sense Discrimination.		**Remarks.**
	Right.	Left.	
G. H.,	9	9	Alcoholism: much tremor.
W. S. W.,	9	9	Do.
J. J.,	8	9	Alcoholic dementia.
W. W.,	8	9	Hypochondriacal melancholia: alcoholism.
B. D.,	6	8	Alcoholism: impulsive insanity.
J. M.,	6	9	Alcoholism: delusional insanity.
W. W.,	7	7	Alcoholism: chronic brain atrophy.
D. F.,	6	6	Alcoholism: delusions of suspicion.
J. W. T.,	4	4	Alcoholism: delusional insanity.
S. B.,	9	9	Adolescent insanity: masturbation.
F. D.,	12	8	Do. do.
T. S. B.,	7	7	Do. do.
J. B. S.,	7	7	Do. do.
W. H. B.,	5	4	Epilepsy: masturbation.
M. C.,	12	5	Right spastic hemiplegia and epileptiform convulsions.
G. S.,	4	5	Old right hemiplegia with early aphasia: epileptiform seizures.
G.E.O.,	5	5	Typical epileptic insanity.

Apparatus for Testing Appreciation of Weight.—The instrument employed for the foregoing observations was made by the Cambridge Scientific Instrument Company, and is described by Francis Galton in the *Journal of the Anthropological Institute*, for May, 1883.

It consists of a box holding ten grooved trays readily movable, each tray supporting three cylindrical weights, all of which are exactly alike in *size and appearance*, differing only in *weight*. The difference in weight between each consecutive pair in a series varies with each tray, the difference being marked on the tray. Commencing with a weight of 1000 grains, a difference of 1 per cent. or 10 grains is the minimum increment adopted; and multiples of this in each series give us a gradually advancing differential from 10 grains up to 120 grains in the tenth tray. Thus, where W = 1000 grains, and the value of $r = 1{\cdot}01$,

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Tray No.	2	has weights	Wr^0	Wr^2	Wr^4
,,	3	,,	Wr^4	Wr^7	Wr^{10}
,,	4	,,	Wr^6	Wr^{10}	Wr^{14}
,,	5	,,	Wr^4	Wr^9	Wr^{14}
,,	6	,,	Wr^0	Wr^6	Wr^{12}
,,	7	,,	Wr^0	Wr^7	Wr^{14}
,,	8	,,	Wr^2	Wr^{10}	Wr^{18}
,,	9	,,	Wr^0	Wr^9	Wr^{18}
,,	10	,,	Wr^4	Wr^{14}	Wr^{24}
,,	12	,,	Wr^0	Wr^{12}	Wr^{24}

Upon the base of each weight the index of the power of r is engraved, and these figures occur also on the side of the tray so as to escape the attention of the person operated upon.

Commencing with a tray where the difference is coarse we gradually work up to the series requiring more delicate appreciation—requesting the person to take the weights between the thumb and forefinger and arrange them in the order of their respective weights. The last series *correctly arranged* gives the minimum differential capacity of the subject. The index of the power of r which occurs on each tray, multiplied by 10 gives us in grains the differential weight appreciated: thus tray 6 gives us a series of three weights differing from each other by 60 grains—*i.e.*, 1000, 1060, 1120—whilst tray 9 differs by 90 grains—*i.e.*, 1000, 1090, 1180, &c.

Reaction-Time.—A large proportion of paralytic subjects are necessarily excluded from attempts at estimating the rapidity of reaction to the stimuli of light and sound; it is only in the earlier stage of the disease, ere the patient has succumbed to any notable degree of dementia, that a reliable record is obtainable. Such results, however, have been secured in the accompanying series of patients, special care having been exercised to exclude any source of fallacy, the result being accepted only after repeated observations, and each record being the average of twenty trials.

REACTION-TIME IN GENERAL PARALYSIS.

		Acoustic Stimulus. Sec.	Optic Stimulus. Sec.
T. P., .	*Maniacal, garrulous, egoistic,*	·16	·25
W. W., .	*Subacute mania, grandiose, noisy, and obtrusive,* .	·17	·24
J. M., .	*Calm, subdued, demented,*	·17	·24
W. L., .	*Wild, maniacal, incoherent, extravagant optimism,*	·18	·18
J. R., .	*Subacute mania, grandiose, and egoistic,* . .	·18	·21
T. S., .	*Tremulous with excitement, optimistic, notable paresis,*	·18	·27
R. C., .	*Calm, notable bulbar paralysis, much optimism,* .	·19	·23
S. M., .	*Calm, dull, heavy, demented,*	·19	·24
J. N., .	*Heavy and demented, depressed, much paresis,* .	·20	·27
C. P., .	*Depressed, obscure egoism, sluggish,* . . .	·21	·27
W. R., .	*Mania, garrulous, obtrusively egoistic,* . .	·22	·23
T. R., .	*Cheerful, calm, slight dementia, no optimism,* .	·24	·30
F. L., .	*Heavy, demented,*	·25	·27

Oculo-motor Symptoms.—The eye-symptoms in general paralysis form a highly characteristic and significant group. Both the extrinsic and intrinsic muscles suffer; but, whilst the former present derangements in exceptional cases only, the latter or intrinsic muscles of the eyeball exhibit deranged innervation, in some way or other, in almost all cases at some stage of the affection.

The motor derangements of the intra-ocular musculature are indicated by—(*a*) size of pupils; (*b*) inequality; (*c*) marginal contour; (*d*) mobility; (*e*) reflex adjustments; (*f*) accommodative adjustments; (*g*) accommodative power. The reflex adjustments (*e*) comprise the pupillary reactions to—(1) cutaneous or sympathetic stimulation; (2) consensual stimulation; (3) direct light stimulation.*

Taking indiscriminately a group of general paralytics in various stages of the disease, the student may meet with one or other of the following pupillary anomalies:—The pupils may be extremely small, perfectly fixed to light, so that on exposing or shading the eye, no movement can be obtained—the pin-hole pupil as it has been called; and it is then said to be in a state of **spastic myosis.** Such a state of contraction is highly important, as being frequently present in general paralysis, locomotor ataxy, and other spinal affections. The pupil may be small, or of moderate size, as the result of paralysis of its dilator or circular fibres; this is called **paralytic myosis,** and may be due to a destructive lesion in the **cilio-spinal** regions of the cord; in this case the pupils no longer dilate with atropine. It is a rare affection, but has been recorded by Baerwinkel in sclerosis of the medulla oblongata.† Unilateral myosis of this description has also been recorded by Nothnagel in disease of the pons. The pupils may be unequal in size, there may be only the slightest degree of inequality, yet if associated with other paretic symptoms, or suspicious mental states, the ocular reflexes should be carefully examined ere the student is prepared to discard such inequality as of trivial import. Care, of course, should be taken to exclude opacities of cornea, capsular adhesions or retinal changes. The inequality may be very extreme from paralysis of one sphincter iridis.

One of both pupils may be in a state of wide dilatation, acting sluggishly, or not at all, to the strongest beam of light—a state of **paralytic mydriasis.** Such a condition may be associated with **amaurosis.**

Or the pupil may be (one or both) irregular in contour; may be oval or not *quite* circular—the upper or lower arc not conforming to the circular outline; here, again, we must carefully exclude adhesions and effects of old iritis. At times the irregularity is very marked and bizarre.

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* It must be understood that the remarks in this chapter apply exclusively to those persistent or gradually progressive impairments of the oculo-motor adjustments, wholly irrespective of those variations in the size of the pupil which may occur from day to day, and which include an *inconstant* factor, such as cortical discharges, &c., or other source of transient stimulus.

† *Journ. Mental Science*, 1878.

Again, the reflex adjustments may fail, and thus upon stimulating the skin by the electric brush, or by a pin, or by pinching the skin, we do not observe the usual dilatation of the pupils in *one*, or perhaps in *either* case; or upon alternately closing one or other eye, the other fails to exhibit the consensual movements of the normal state; and this, likewise, may be observed in one eye only or in both. In a state of *spastic myosis*, of course, both the foregoing reactions are abolished. "In the healthy eye the consensual contraction, according to Listing, does not begin until two-fifths of a second after the opening of the other eye, and lasts about one-fifth of a second, after which the pupil again dilates slowly, and vibrates for some seconds. The consensual dilatation he observed to commence about half a second after the closing of the other eye, and with diminishing rapidity to continue for one or two seconds."* Then again, the sphincter iridis, either when the pupils are equal or very dissimilar in size, may not respond to the stimulus of *light*, or may respond with a sluggishness evidently morbid. This condition of failure of the **light-reflex** without a similar implication of the accommodative movements of the iris is called **reflex iridoplegia,** or the Argyll-Robertson symptom, which is one of great significance in early stages of tabes and general paralysis. Yet again, the sphincter may show no response to light, nor to the effort of accommodation, nor the movements of convergence and divergence; and the resulting paralysis we speak of as an **associative iridoplegia.**

Finally, accommodation itself may be impaired or lost in one or in both eyes from paralysis of the ciliary muscle (tensor choroideæ), and, if this be associated with paralysis of the sphincter iridis to light and to convergence, and of the dilating mechanism of the pupil, we may speak of the condition as one of **cycloplegic iridoplegia,** or use Mr. Hutchinson's term, **ophthalmoplegia interna.**

Any one of these numerous anomalies may present themselves in the subjects of general paralysis. The contraction which occurs during accommodation for a near object, and when the eyeballs are convergent, must be regarded as of the nature of an *associated movement*; yet we must not understand by this that the accommodative movement is involuntary. "The fact that this last (contraction during accommodation) is only an associated movement, does not deprive it of its voluntary character."†

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* Quoted by Donders, "Accommodation and Refraction of the Eye," *Syd. Soc.*, p. 573.

† Donders, *loc. cit.*, p. 574.

The more frequent motor derangements met with may thus be summarised :—

Consensual paralysis.	(1) Loss of consensual movements.
Partial reflex iridoplegia.	(2) Loss of light reflex.
	(3) Loss of skin-reflex or of reflex dilatation.
Complete reflex iridoplegia.	(4) Both the above conjoined.
Associative iridoplegia.	(5) Loss of contraction upon convergence only.
Complete iridoplegia.	(6) All the above conjoined.
Cycloplegic iridoplegia.	(7) Loss of accommodation superadded.

As to the relative frequency with which these derangements to the irido-muscular apparatus occurs, we usually find as an early sign a slight, perhaps scarcely appreciable, inequality of the pupils, the sizes of which are otherwise not abnormal, accompanied by a little sluggish delay on the part of the larger in reacting to light, while the smaller contracts and dilates briskly. If the light be bright this want of active mobility may not be appreciable, hence the necessity of testing in a subdued light as well as by focal illumination. If the patient be now told to converge the eyeballs, the pupils contract readily and equably, and we regard the case as one of commencing *reflex iridoplegia.* If this be the case, we shall now almost certainly find associated with it, the loss of the sympathetic dilatation which should occur on irritating the skin; for this is, of all other iridal paralyses, the earliest observed. The strong stimulation of a sensory nerve is well known to inhibit reflex actions; and upon this physiological principle, Bechterew would explain this pupillary dilatation as, in fact, an *inhibition of the usual light-reflex;* it is equally produced by noises in the ear, or by stimulation of the sexual organs, uterine pain, &c. The constant association of these two anomalous states is readily explained by the proximity of the sympathetic tract supplying the *dilator iridis* to that nucleus of the oculo-motor which regulates the sphincter iridis under the stimulation of light.

The large proportion of paralytics who present themselves in an early stage will afford us these signs—viz., a moderate-sized pupil, slightly larger than its fellow, sluggishly reacting to light, even to a bright beam, and absence of the sympathetic dilatation.

In the *more advanced stages,* the larger pupil will now be found *quite fixed to light* or may contract very partially; and, if a strong beam of light be used to illuminate the eye, the initial slight contraction is followed by a sudden dilatation beyond its original limits; remaining wide throughout the illumination of the retina. One eye succumbs to this reflex iridoplegia before the other; but, we often recognise a failing mobility in the healthier organ also, and eventually both become quite fixed and immobile to light. The small-sized pupil (*myosis*), although usually noted at an early stage of the disease, is not thus restricted; it may retain this size throughout the disease, and be a notable sign even to the fatal termination. On the other hand, *mydriasis*, if not associated with distinct *amaurosis*, is a feature of the later stage of general paralysis. The student would do well, when examining the eye of a presumed case of general paralysis, not only to measure with the pupilometer, but also to compare the dimensions with the healthy eyes of those standing by, *under the same intensity of light*. When testing the response to shading he should not rest satisfied with the effect produced by covering one eye only, but should note the much greater range of dilatation obtained when *both* are shaded. He should also, when testing the light reflex, be careful to exclude accommodative efforts. As the small pupil in the early days of general paralysis becomes gradually larger with advancing reflex iridoplegia, it affords us evidence of a deeper implication of the nuclei in one half of the pons, *as well as of the cerebral hemisphere of the same side;* for it has constantly occurred to the writer to observe that when unilateral convulsions or paralysis occur in the early stages of general paralysis, the dilated pupil is *on the side of the discharging or paralysing lesion.** It appears to us unquestionable that the oculo-motor disturbances, which we have above alluded to, are greater on the side of the more deeply-implicated hemisphere.

The *pinhead pupil* may persist to the end, and yet present no impairment of the associated movements on accommodation, as in the following cases :—

	Size of Pupils.	Reaction to Light.	Sympathetic Reflex.	Consensual Reflex.	Accommodation.
D. R.,	$\frac{1{\cdot}75}{1{\cdot}5}$ millimetres.	Immobile.	Immobile.	Immobile.	Normal and active.
S. A. L.,	$\frac{1{\cdot}5}{1{\cdot}5}$ millimetres.	Immobile.	Immobile.	Slight in left only.	Brisk.

In estimating the significance of these oculo-motor anomalies, the student must bear in mind the teaching of experimental physiology upon the subject, which demonstrates

(1) That centripetal retinal excitations travel by way of the optic nerve and tracts to the upper quadrigeminal arc—*i.e.*, by the *nates*, its *brachia*, the *external geniculate*, and the *pulvinar* of the *optic thalamus*, which constitute a first stage or level, and from which such excitations pass by the optic radiations to the occipito-angular region, or visual centre of Ferrier.

(2) That section of one optic nerve causes monocular blindness, together with loss of the light-reflex (*reflex iridoplegia*) of the same eye, still with persistent contraction of both eyes on stimulation of the *second eye*—a phenomenon explained by the coupling of the oculo-motor nuclei.

(3) That section of one optic tract issues in *homonymous hemiopia*, from paralysis of the corresponding retinal halves of both eyes. Whilst Bechterew shows

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* See Article by Author in *West Riding Asylum Reports*, vol. vi.

that, in the dog, division of one tract does not affect the reflex contraction of the constrictor iridis, Knoll indicates that in the rabbit (with its complete decussation at the *chiasma*) division of one tract abolishes the light-reflex in the *opposite* eye.

(4) That enucleation of one eyeball in animals with fairly complete decussation at chiasma (rabbit) issues in atrophy of the *nates*, its *brachia*, and *external geniculate*, with the *pulvinar*, all of the *opposite* side; that in animals with very incomplete decussation—*i.e.*, where the direct fibres preponderate (*Erb* and *Day*), such atrophy pertains more equally to these parts on *both* sides. The representation of the retinal fields in these ganglionic centra behind the optic commissure will vary with the animal; and so lesions of the tract or of the *quadrigeminal* body of one side will issue in varying results. Thus Baumgarten [*] records secondary degeneration of *both optic tracts in man* after destruction of *one eye.*

In this lower arc, therefore, connecting the retina with the mesencephalic centres, we find that section and destructive lesions on the peripheral side of the ganglia issue in—

Partial or complete amaurosis, with partial loss of the consensual reflex and reflex-iridoplegia.

Homonymous hemiopia, with, or without, impairment of light-reflex; certain secondary degenerative changes affecting the tracts and the quadrigeminal structures with the pulvinar alluded to above..

In like manner, destruction of these ganglia also issues in degenerative changes in both optic nerves, especially that opposite the lesion. *Such degenerations are limited to this arc, and do not travel centrally beyond the quadrigeminal centres to the cerebral cortex.*

On the other hand, the upper arc of optic radiations is connected with the visual perceptive centres of the cortex. Its division is followed by atrophy of the cortex and of the quadrigeminal arc, as well as of the lower arc connected with the retina; but it must be remembered that the retinal reflexes are not abolished by lesion *above the mesencephalon.*

Size of Pupils.—The pupillary aperture is more frequently found dilated than unduly small and constricted, and a moderate-sized pupil is less frequent than one distinctly larger than usual. In fact, if we take note of all cases of unilateral *mydriasis*, as well as of those wherein both are dilated, we shall find that it is met with in one-half of our cases. If we arbitrarily assume any size up to 2 millimetres diameter to include the *small or contracted pupil*, from above 2 millimetres to 3 millimetres for the *moderate-sized pupil*, and all above 3 millimetres as *large dilated pupils*, we get the following proportions:—

Small contracted pupil, ...	4	cases.
Moderate-sized, ...	13	,,
Dilated, ...	27	,,
	44	,,

In about half the cases—*i.e.*, in twenty-one—one or both pupils measured 4 millimetres or upwards, and in six of these cases *both* pupils were equal in size. *Very large pupils*—6 to 7 millimetres—prevailed in three cases (see Summary, pp. 309–313). In the Summary, the upper figure of the fraction in col. 2, and corresponding line in col. 3, indicate in each case the size of pupillary aperture and iridal reactions of the *right eye;* the lower figure and line refer to the same features in the *left eye.* In all other cases the reactions are alike for both eyes.

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[*] *Central-blatt. f. d. Med. Wissenschaft*, 1883. (Quoted by *Ferrier.*)

Unilateral deviations were noted in twenty-seven cases, the remaining seventeen having pupils of equal dimensions. In sixteen cases the right, and in eleven cases the left, was the larger pupil of the two. We have endeavoured to show elsewhere that an important distinction must be made between *early* and *late* inequality of pupils in general paralysis, or, rather, between the inequality not associated with impairment of the pupillary reflexes, and those cases where unequal pupils manifest also the absence of reaction to light and to other stimuli.* The former, or earlier, sign is an indication of the cortical lesions established; whilst the latter, or defective iridal reaction, is due to advancing lesions of *spinal* and *bulbar* arrangements.

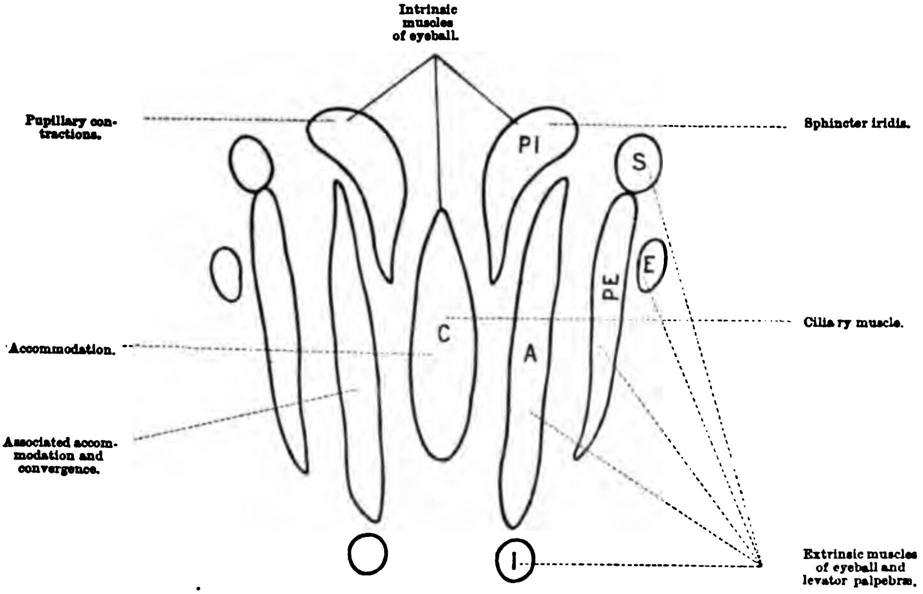
Intrinsic muscles of eyeball.
Pupillary contractions.
Sphincter iridis.
PI
S
E
PE
C
A
Ciliary muscle.
Accommodation.
Associated accommodation and convergence.
I
Extrinsic muscles of eyeball and levator palpebræ.

Fig. 19.—Scheme of segmentation of third nerve nuclei showing presumed anatomical and physiological relationships.

A, Anterior nucleus; *C*, Central or median nucleus; *E*, External nucleus of Bruce; *I*, Inferior nucleus (segment of anterior); *S*, Superior or nucleus of Darkschewitsch; *PI*, Postero-internal or pale nucleus of Edinger-Westphal; *PE*, Postero-external or postero-lateral nucleus.

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* "Remarks on Ocular Symptoms of General Paralysis of the Insane, with special reference to its Clinical Groupings," *Brit. Med. Journ.*, April and May, 1896.

ANALYSIS OF FORTY-FOUR CASES OF GENERAL PARALYSIS.

Pupillary Reaction.	Light-Reflex.		Focal Illumination.		Consensual.		Reflex Dilatation.		Associated Movement.		Superficial and Deep Reflexes.	Patella-Reflex.		Plantar-Reflex.	
		Per cent.		Per cent.		Per cent.		Per cent.		Per cent.			Per cent.		Per cent.
Both Pupils Fixed, . .	16	36·3	15	34	19	43·1	28	63·6	5	11·3	Both Absent.	7	15·9	3	6·8
,, ,, Sluggish, .	2	4·5	...	...	2	4·5	...	...	2	4·5	Nearly Absent.	2	4·5	...	...
,, Extremely ,, .	5	11·3	1	2·2	1	2·2	2	4·5	2	4·5	Sluggish.	1	2·2	7	15·9
,, Slight Contr., . .	5	11·3	4	9	4	9	2	4·5	...	...	Very Sluggish.	1	2·2	2	4·5
Normal, ...	8	18·2	7	15·9	11	25	5	11·3	28	63·6	Both Normal.	6	13·6	19	43·1
Different, ...	8	18·2	10	22·7	6	13·6	6	13·6	6	13·6	,, Different.	9	20·4	4	9
Active, but Limited, Oscillates and Dilates, .	...	...	6	13·6	1	2·2	...	...	...	...	,, Exaggerated.	16	36·3	8	18·2
Doubtful, ...	...	...	...	...	...	...	1	2·2	1	2·2	Doubtful.	2	4·5	1	2·2
Extremely Limited Range,	...	...	1	2·2	...	...	...	...	...	...	...	...	...	...	...

Right Pupil Largest, ...	16
Left ,, ,, ...	11
Pupils of Equal Size, ...	17
	44

SUMMARY OF OCULO-MOTOR AND ASSOCIATED ANOMALIES IN

	Size in mm.	Light-Reflex.	Focal Illumination.	Consensual.	Reflex Dilatation.	Associated Movement.
J. W. M.,	$\frac{4\frac{3}{4}}{3}$	Almost fixed. Slight contn.	Slight contn.	Normal.	Fixed.	Normal.
J. O. O.,	$\frac{3}{4}$	Slight. Normal.	Slight. Normal.	„	„	„
M. P., .	$\frac{2\frac{3}{4}}{4}$	Normal.	Normal.	„	„	„
A. S., .	$\frac{3\frac{1}{2}}{4\frac{1}{2}}$	„	„	Sluggish.	„	Sluggish.
J. D., .	$\frac{4}{3\frac{1}{4}}$	Sluggish.	Slight, then oscillates. 2dy dilatation.	Normal.	Normal.	Normal.
M. B., .	$\frac{3}{3}$	Normal.	Normal.	„	Fixed.	„
L. B., .	$\frac{3\frac{1}{4}}{2\frac{1}{4}}$	„	Normal for both. 2dy dilatation.	„	Normal.	„
F. S., .	$\frac{4}{4}$	„	Normal.	„	Normal. Sluggish.	„
K. T., .	$\frac{3\frac{1}{4}}{3}$	„	„	Normal. Dilates.	Sluggish.	„
S. S., .	$\frac{2}{2}$	„	„	Normal.	Normal.	„
J. P., .	$\frac{3\frac{1}{4}}{3\frac{1}{4}}$	„	„	„	Fixed.	„
J. W., .	$\frac{3}{3}$	Sluggish.	Normal, then oscillates 2dy dilatation.	„	Normal.	Normal. Sluggish.
J. L., .	$\frac{2\frac{1}{2}}{3}$	Sluggish and of limited range.	Normal but of limited range.	„	Slight.	Normal.
R. S., .	$\frac{2\frac{1}{2}}{2\frac{1}{2}}$	Fixed. Slight.	Slight. Normal.	„	Normal.	„
A. H., .	$\frac{2}{3}$	Fixed.	Fixed. Sluggish.	Fixed. Slight.	Fixed. Slight.	Fixed. Slight.
S. A. L.,	$\frac{1{\cdot}5}{1{\cdot}5}$	„	Fixed.	Fixed. Slight.	Fixed.	Normal.
D. R., .	$\frac{1\frac{3}{4}}{1\frac{1}{2}}$	„	„	Fixed.	„	„
J. W., .	$\frac{1\frac{3}{4}}{1\frac{3}{4}}$	„	Fixed or of extremely limited range.	„	„	Extremely sluggish and limited.
G. B., .	$\frac{2}{2\frac{1}{4}}$	„	Fixed.	„	„	Normal.
T. W. H.,	$\frac{2\frac{3}{4}}{3}$	„	„	„	„	„
J. C., .	$\frac{2\frac{1}{2}}{2\frac{1}{2}}$	„	„	„	„	„

FORTY-FOUR CASES OF GENERAL PARALYSIS.

Accommodation.	Visual Acuity.	Colour Sense.	Patella-Reflex.	Plantar-Reflex.	Gait, &c.
J. $\frac{4}{23}$ and $\frac{1}{23}$	Sn. $\frac{5}{6}$	Normal.	Normal.	Normal.	Brisk.
J. $\frac{1}{22}$ Sn. $\frac{\cdot 5}{24}$	Sn. $\frac{6}{10}$	,,	Exag.	,,	Brisk.
J. $\frac{3}{22}$ Sn. $\frac{\cdot 5}{22}$	Sn. $\frac{6}{10}$	,,	,,	,,	Brisk.
J. $\frac{1}{21}$ Sn. $\frac{\cdot 5}{23}$	Sn. $\frac{6}{6}$	,,	Slightly Exag.	,,	Brisk, trunk stiff.
J. No. 4	Sn. $\frac{6}{10}$ Sn. $\frac{6}{20}$	,,	Sluggish.	,,	Brisk.
J. $\frac{1}{18}$	Sn. $\frac{5}{6}$ Sn. $\frac{6}{10}$	,,	Normal.	Sluggish.	Brisk.
J. No. 4	Sn. $\frac{6}{10}$	,,	,,	,,	Brisk.
J. No. 2 Sn. $\frac{\cdot 5}{27}$	Sn. $\frac{6}{12}$	Normal but green = blue.	Sluggish.	Normal.	Brisk, and runs.
? dil.	Sn. $\frac{6}{10}$	Normal.	Almost absent.	Absent.	Sways, staggers, and leans to right.
J. $\frac{6}{16}$	Sn. $\frac{6}{30}$	,,	Exag.	Exag.	Stiff, tottering, feeble.
J. $\frac{6}{25}$	Sn. $\frac{6}{10}$	Normal but b. = puce, y. = pink.	,,	,,	Brisk, but stiff, a little tottering.
J. $\frac{6}{28}$	Sn. $\frac{6}{10}$	Normal but b. = green.	,,	Normal.	Stooping, staggering, and falls if eyes be closed.
J. No. 3 Sn. $\frac{\cdot 5}{25}$	Sn. $\frac{6}{12}$	y. = orange, g. = puce.	,,	,,	Brisk.
?	?	?	,, Normal.	,,	Right leg stiff—not dragged = right hemiplegia c. aphasia, horizontal nystagmus.
Sn. $\frac{\cdot 6}{34}$	Sn. $\frac{6}{6}$	Normal.	Absent.	,,	Stiff, slow, sways.
?	?	,,	,,	Exag.	Brisk, elastic.
J. 1 Sn. $\frac{\cdot 5}{25}$	Sn. $\frac{6}{5}$	Normal but green, more like blue than yellow.	Sluggish. Normal.	Absent.	Brisk and springy.
Sn. $\frac{?6}{30}$	Sn. $\frac{6}{10}$	r. = pink, y. = purple, b. & green = Normal.	Exag. Normal.	Exag. Normal.	Slow, laboured, leans to right side.
J. No. 1 Sn. $\frac{\cdot 5}{20}$	Sn. $\frac{6}{6}$	Normal.	Sl. exag.	Normal Sluggish.	Brisk.
J. $\frac{1}{20}$	Sn. $\frac{5}{6}$	,,	Slight. Absent.	Normal.	Stiff.
Sn. $\frac{2\frac{1}{4}}{20}$	Sn. $\frac{4}{30}$	Normal but green = yellow.	Absent.	,,	Stiff.

	Size in mm.	Light-Reflex.	Focal Illumination.	Consensual.	Reflex Dilatation.	Associated Movement.
J. H. W.,	$\frac{2\frac{3}{4}}{2\frac{1}{4}}$	Fixed.	Fixed.	Slight. Fixed.	Fixed.	Normal.
B. K., .	$\frac{3\frac{1}{2}}{3\frac{1}{4}}$	,,	,,	Fixed.	,,	Fixed.
W. M., .	$\frac{3}{3}$	,,	,,	,,	,,	,,
C. J. C.,.	$\frac{4\frac{1}{4}}{4\frac{1}{2}}$	,,	,,	,,	,,	Fixed. Normal.
J. J. .	$\frac{4\frac{1}{2}}{4}$	,,	,,	,,	,,	Sluggish.
J. H., .	$\frac{4}{3}$	,,	,,	,,	,,	Fixed. Normal.
J. L., .	$\frac{4\frac{1}{2}}{4\frac{1}{2}}$	,,	,,	,,	,,	Fixed.
J. C. C.,.	$\frac{6\frac{1}{2}}{6}$	,,	,,	,,	,,	,,
J. M., .	$\frac{7}{5}$	,,	,,	,,	,,	,,
J. A., .	$\frac{3\frac{1}{2}}{4\frac{3}{4}}$	Slight, 2^{dy} dilatation. Fixed.	,,	,,	,,	? dil.
E. B., .	$\frac{2\frac{1}{4}}{1\frac{3}{4}}$	Fixed. Slight, 2^{dy} dilatation.	Fixed. Slight, 2^{dy} dilatation.	,,	,,	Normal.
J. H., .	$\frac{3\frac{1}{2}}{3\frac{1}{2}}$	Sluggish.	Sluggish. 2^{dy} dilatation.	Slight.	Normal. Slight.	,,
A. S., .	$\frac{4}{4}$	Normal. Sluggish.	Normal. Sluggish, 2^{dy} dilatation.	Fixed.	Fixed.	,,
J. T., .	$\frac{4}{4\frac{1}{4}}$	Sluggish and of limited range.	Sluggish. Sluggish.	,,	Fixed. Sluggish.	,,
T. T., .	$\frac{4}{4}$	Sluggish and of limited range.	Normal. Sluggish.	Sluggish.	Slight.	,,
C. G., .	$\frac{6}{6}$	Sluggish.	Normal with 2^{dy} dilatation.	,,	Sluggish. Fixed.	Sluggish.
J. M., .	$\frac{2\frac{1}{4}}{2}$	Slight with 2^{dy} dilatation.	Slight with 2^{dy} dilatation.	Fixed.	Fixed.	Normal.
J. B., .	$\frac{3\frac{1}{4}}{3\frac{1}{4}}$	,,	Normal with 2^{dy} dilatation.	,,	,,	,,
W. T. S.,	$\frac{4\frac{1}{2}}{4\frac{1}{4}}$	Sluggish. Sluggish.	Normal Sluggish, 2^{dy} dilatation.	Normal. Sluggish.	Sluggish.	Normal. Sluggish.
W. S., .	$\frac{4\frac{1}{4}}{3\frac{3}{4}}$	Sluggish.	Normal, then oscillates with 2^{dy} dilatation.	Sluggish.	Sluggish. Fixed.	Normal.
H. M., .	$\frac{3}{2\frac{3}{4}}$	Sluggish. Fixed.	Normal. Fixed.	Normal. Fixed.	Fixed.	Slight. Fixed.
B. H., .	$\frac{4}{3}$	Slight.	Slight.	Slight.	,,	Normal.
J. B., .	$\frac{4\frac{1}{2}}{4\frac{1}{2}}$	Slight with 2^{dy} dilatation.	Slight, 2^{dy} dilatation. Normal, 2^{dy} dilatation.	,,	?	,,

Note.—The upper figure of the fraction in col. 2 and corresponding line indicate in each case the size
same features in the *left eye*. In all other

Accommodation.	Visual Acuity.	Colour Sense.	Patella-Reflex.	Plantar-Reflex.	Gait, &c.
J. $\frac{1}{26}$	Sn. $\frac{5}{6}$	Normal.	Normal.	Sluggish.	Stiff, broad basis, heels down first.
?	Sn. 50 only	,,	Exag.	,,	Legs drop, left hemiplegia = contracture of left arm, cannot stand unsupported.
Sn. $\frac{\cdot5}{20}$ Sn. $\frac{\cdot6}{20}$	Sn. $\frac{6}{6}$ Sn. $\frac{6}{12}$	,,	?	Normal.	Brisk.
?	Sn. $\frac{6}{15}$	,,	Normal. Exag.	Absent.	Leans to left side.
J. No. 1 Sn. ·5	Sn. $\frac{6}{6}$	Normal but gr. = ?	,,	Normal.	Stiff.
J. $\frac{14}{25}$ Sn. $\frac{3}{25}$	Sn. $\frac{6}{60}$ Sn. $\frac{}{6}$	Normal.	Absent.	Exag.	Brisk.
?	?	Normal but y. = ?	Exag. clonos.	Sluggish.	Quick, elastic. Both lenses opalescent.
?	Sn. $\frac{6}{6}$	Normal.	Almost nil.	Normal.	Brisk.
J. No. 2	?	Normal but gr. = yellow.	Exag.	Sluggish.	Flexion and rigidity of both legs and right elbow; cannot stand. Adhesions in both, pigmentary deposit on front of lens.
?	?	?	Absent.	Normal.	Stiff, waddling, but stands unsupported.
J. $\frac{4}{24}$	Sn. $\frac{6}{10}$	Normal.	Exag.	Slight.	Stiff.
? dilatn.	?	?	Normal.	Sluggish.	Heavy, tottering.
Sn. $\frac{14}{27}$	Sn. $\frac{6}{18}$	Normal but gr. = crimson.	Sluggish. Normal.	Exag.	Stooping, bent, unsteady, insecure.
? dilatn.	?	?	Exag.	Sluggish.	Left eye strongly convergent when eyes are directed to right.
Sn. $\frac{\cdot5}{17}$	Sn. $\frac{6}{24}$	Normal.	Absent.	Normal.	Brisk.
Sn. $\frac{2}{18}$	Sn. $\frac{6}{40}$	Normal but gr. = puce.	Normal.	,,	Brisk.
J. $\frac{1}{19}$	Sn. $\frac{6}{10}$	Normal.	Exag.	,,	Brisk.
J. $\frac{5}{21}$	Sn. $\frac{6}{10}$	,,	Exag. Absent.	Sluggish. Normal.	Right leg dragged; right arm contracted; feeble grasp.
J. No. 1	Sn. $\frac{\cdot5}{25}$	,,	Absent.	Exag.	Brisk.
J. No. 4	Sn. $\frac{6}{30}$	,,	Sluggish.	Normal.	Brisk, springy.
?	?	?	?	?	Stooping, shuffling, most insecure.
J. $\frac{5}{42}$	Sn. $\frac{5}{6}$ Sn. $\frac{6}{6}$	Normal.	Exag.	Normal.	Stiff.
?	?	?	,,	Sluggish.	Cannot walk or stand.

of pupillary aperture, and iridal reactions of the *right eye:* the lower figure and line **refer to the cases** the reactions are alike for both eyes.

Light-reflex.—Referring to our table of actual figures, it is found that over 36 per cent. have *both* pupils *perfectly immobile* and fixed to the stimulus of light, and that half as many—*i.e.*, 18 per cent.—show fixity or sluggish reaction in one or other eye. Further, in 11 per cent. both pupils were noted as *excessively sluggish* in reaction and limited in their range, and in 18 per cent. only could it be stated that the pupils reacted *normally* under the stimulus of light. The immobility of the pupils is rigid even to focal illumination of the eye by a convex lens, and with a strong light—as many as 34 per cent. still exhibiting both pupils immobile.

An early indication of commencing iridoplegia is given by focal illumination, for, as shown by the table, 13·6 per cent., although active to light, show (for a concentrated beam of light) a most limited range of movement, together with an oscillation which then tends to *wide dilatation even under this bright illumination of the retina.* This tendency to *dilate during stimulation by light* appears to me to be the earliest augury of coming paralysis.

Consensual Movements.—When one eye is alternately shaded and exposed, the accommodation being relaxed, we observe that both pupils dilate and then contract. We speak of this reaction in the eye not shaded as the **consensual reflex** (dilatation or contraction); and of the reaction in the eye alternately shaded and exposed, as the **direct light reflex.** As to the course taken by the stimulus for these consensual movements, it is a well-known fact that lesions of one optic tract, resulting in homonymous hemianopsia, do not affect the consensual reaction (*Knoll*, *Bechterew*, *Baumgarten*, *Erb*); and it follows from this that the stimuli pass back not along the crossed fibres of the chiasma alone, but also by the alternative route of direct fibres of the optic tract (see diagram, p. 318). In testing the consensual and direct reaction it is well to bear in mind that the consensual and direct reflex dilatations are *equal in* normal eyes; hence the shaded and exposed eye will exhibit equally dilated pupils, since the covered eye lessens the contraction of the exposed eye to just the same degree as the exposure of the latter diminishes the dilatation of the former. It is on this account that we should test the energy of dilatation by shading *both* eyes (p. 305).

These reactions were abolished in 43 per cent. of the total cases, and were almost invariably absent where the light-reflex was absent in *both pupils.* A considerable number, however, of cases of incomplete or commencing paralysis to light showed perfectly normal consensual movements (25 per cent.), or but slight impairment, amounting to sluggish response or unequal response, on both sides.

The failure of the consensual movements apparently never occurs apart from impairment of the direct or light-reflex (the only exception, if it is one at all, is that of *K. T.*, where *very sluggish* dilatation is noted with normal light-reflex). It appears invariably to follow upon the latter impairment, and thus we find in a few cases (*R. S.*, *e.g.*), that the light-reflex is impaired, whilst the consensual activity is normal in both eyes.

Reflex Dilatation (*Erb*).—Every acoustic, tactile, painful or electric stimulus will entail a dilated pupil, and the excitation of the skin by pricking, pinching, or by electric brush, is the usual method adopted for judging of the integrity of this reflex dilatation. It must be remembered that actual sensation need not follow upon this stimulation and its resulting dilatation; thus, stimulation of the anæsthetic skin of the hemianæsthetic subject will induce pupillary dilatation just as readily as can be done in cases of coma and sleep.

This movement, which, as before stated, Bechterew regards in the light of an inhibitory action, fails at an early date. It was completely abolished in 63·6 per cent., and normal response was obtained only in 11·3 per cent. Excitation of any available sensory surface alike fails to produce response in such cases; and, it is of interest to note, that unilateral failure of this reflex dilatation also occurs (being present in 13·6 per cent.). In several of the cases tabulated, it will be noted that this reflex dilatation failed where the pupils showed healthy and active response to the stimulus of light, both directly and consensually (see *U. P.; A. S.; M. B.; K. T.;* and *J. P.*). This anomalous condition we believe to be the earliest sign of approaching irido-motor implication; following in its wake comes the sluggish reaction of one pupil to light with a tendency to dilate on sustained illumination; then a gradually extending *paralytic mydriasis*, with which becomes associated the impairment of consensual activity.

Associated Irido-Motor States.—The associated movements of contraction and dilatation of the pupil during the act of accommodation and efforts of convergence are affected only in the later stages of the disease; and in five cases only (or 11·3 per cent.) was it absolutely lost in both eyes; whilst, as many as twenty-eight (or 63·6 per cent.) showed perfectly-normal response. It may likewise show unilateral impairment or abolition; and in several cases (*F. B.; C. J. C.; A. H.;* and *J. W.*) with complete abolition of the light-reflex in *both eyes*, the associated iridoplegia appeared but on one side, the other pupil acting vigorously to convergent efforts.* So far from associative iridoplegia (*i.e.*, loss of pupillary contraction upon convergence) being the invariable sequence to the Argyll-Robertson pupil, the fact seems e that this latter symptom remains almost invariably persistent up the fatal issue; whilst loss of accommodative movements (associative plegia), when present, is found from the first. The fact that ure of response to light, on convergence, and on shading runs ally on to failure of accommodation also (cycloplegia), is explained Mr. Hutchinson as possibly due in tabetic subjects to implication e ciliary ganglion, which forms a crossway for all the nerve-fibres d.* But we are of opinion that in progressive paralysis the site on is more often nuclear, the degeneration involving the of the motor-oculi nucleus and some as yet unascertained n of this nucleus with the vaso-motor.†

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* It has been conclusively shown by Donders, as well as by De Ruiter and Cramer, that the associated contraction of the pupil occurs with the act of accommodation, when there is no increased convergence of the visual line, and also with the latter when there is no change in accommodation. *Loc. cit.*, p. 574.

m the study of a large number of cases of paralytics showing se oculo-motor troubles, it appears to us that—

Firstly, the smaller pupil is upon the side of lesion of the oculomotor nucleus, or the larger pupil is opposite to the nucleus involved.

Secondly, that the smaller pupil is the one which fails to act *consensually*, if one only shows a failure in this respect.

Thirdly, that the smaller pupil is the one in which light-reflex is most impaired or is abolished, if both are not equally implicated in this respect.

If it be accepted that the path of the light-reflex is through the central decussating fibres of the chiasma to the opposite oculo-motor nucleus (the decussating opto-geniculate tracts), as well as to the constrictor centre of the same side by means of the intercentral link, then a lesion of one centre, say the *right* motor-oculi, will intercept the path of stimuli between the left retinal field and *both* irides. But although the left eye can pass no stimuli to either *constrictor pupillæ*, its iris can still be affected by stimulation of the opposite eye, through the crossed opto-geniculate tract. The left pupil, therefore, as well as the right, would be paralysed or fixed to direct light stimulation; but whilst the left would still be consensually affected, the right would be fixed to both influences. The six cases afforded by our series confirm these views in every respect.

It thus appears that there are two links betwixt the quadrigeminal centres and the cortex, on the one hand, and the periphery on the other. An upper cortical and a lower or retinal—consisting respectively of the optic radiations from the thalamus and mesencephalon, extending to the occipito-angular region; and a lower link from the retina to the mesencephalon; the upper being essential for visual perceptions—the lower being also the centripetal paths for the irido-motor reflexes. Lesions of the lower retinal link are productive of secondary changes backwards to the quadrigeminal regions (Obs. 4, p. 306), but not beyond this limit; lesions of the upper or cortical link cause degenerations, which spread both centrally and peripherically, involving both cortex, optic tracts, and the intervening ganglionic centre. The immediate result of a lesion of one of the lower links is impairment of vision—either complete amaurosis or paralysis of the associated retinal fields (*equatorial* or *homonymous hemiopia*), the irido-motor reflexes being involved only when the lesion is on the peripheral side of the chiasma. Since, however, the escape of the irido-motor reflexes depends (in this case, when the nuclei in the medulla are intact) upon the commissural connections, incomplete implication of both tracts must necessarily result not only in visual, but also in reflex-iridal, disturbances. On the other hand, the cortical, or upper link, when first implicated, has visual disturbances only for its symptoms, for the iridal reflexes are not involved; and thus complete blindness with still active pupils indicates a blindness due to *cortical lesion* or one *beyond* the quadrigeminal bodies (this condition as a functional disturbance occurs—*e.g.*, in *uræmic* poisoning). Eventually, however, the consecutive degeneration passing to the upper *quadrigeminal* and *external geniculate* bodies, &c., leads also to disturbances of irido-motor reaction. The individual nuclei, defined by Henson and Voelcker as extending in front of the aqueduct, may, however, be picked out by morbid processes; and, in this case, the iridal reactions suffer without any necessary implication of vision, a condition frequently seen in the early stages of general paralysis.

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* "On Paralysis of the Internal Muscles of the Eye" (Ophthalmoplegia Interna), *Medico-Chir. Trans.*, vol. xi., p. 216.

† For a further discussion on this point see article by author, *loc. cit.*

Significance of Certain Pupillary Anomalies.—The true sign of a paralysis of the cervical sympathetic trunk, or its centre in the cord, is not so much a *small* pupil, as the failure to dilate upon shading the eye. Mr. Jonathan Hutchinson has shown that cases of such sympathetic paralysis occasionally occur with a *moderate-sized* pupil.*

It is customary, however, to regard the myosis of tabetic cases as due to implication of the posterior columns of the spinal cord; whilst the failure to contract with light, the associated contraction on convergence being retained, is explained by implication of Meynert's fibres connecting the optic fibres with the motor oculi nucleus (see Fig. 20).

Now, if it be recalled that this partial reflex iridoplegia is frequently *unilateral*, we see at once the difficulty of admitting this explanation as correct. *Bilateral* implication of Meynert's fibres would certainly lead to double reflex iridoplegia; but, since the third nerve nuclei are regarded as tied together by commissural fibres, implication of one side could not possibly affect the reaction to light of either pupil. Latterly, I have been accustomed to explain away this difficulty by suggesting a hypothetical tract of fibres to exist (see Fig. 20), connecting the third nerve nuclei with the dilator centre of the sympathetic in the medulla. In fact, we may regard every retinal stimulation by *light* as also *inhibiting* the dilating centre in the medulla (through these hypothetical fibres—see diagram), these fibres being the tract whereby the sympathetic is influenced by differential light stimuli alone, all other stimuli, as from the cutaneous surfaces, passing through the usual course in the posterior columns of the cord direct to the dilator centre in the medulla.

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* "Notes on the Symptom Significance of Different States of the Pupil," *Brain*, vol. i., p. 11.

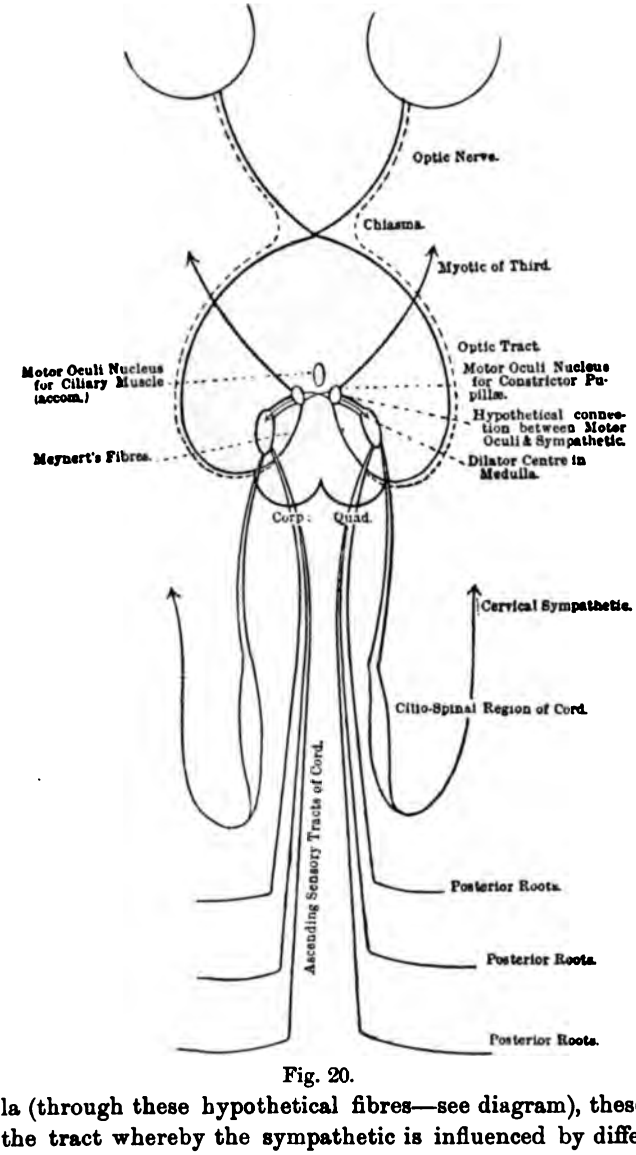
Optic Nerve.
Chiasma.
Myotic of Third.
Optic Tract.
Motor Oculi Nucleus for Ciliary Muscle (accom.)
Motor Oculi Nucleus for Constrictor Pupillæ.
Hypothetical connection between Motor Oculi & Sympathetic.
Meynert's Fibres.
Dilator Centre in Medulla.
Corp: Quad.
Cervical Sympathetic.
Cilio-Spinal Region of Cord.
Ascending Sensory Tracts of Cord.
Posterior Roots.
Posterior Roots.
Posterior Roots.

Fig. 20.

Shading the eye, therefore, lessens the inhibitory effect of retinal stimuli, and the dilator centre strongly functionates; division of these hypothetical fibres would issue in a *failure to dilate on shading*, with, probably, a moderate-sized pupil, whilst cutaneous and other forms of sensory stimuli still effect pupillary dilatation; extensive disease of the sensory columns of the cord would impair or destroy pupillary *dilatation to cutaneous stimuli*, yet *not to shading*; whilst disease of the dilator centre in the medulla, or the cilio-spinal region of the cord, would abolish dilatation of the pupil for all these forms of sensory stimulation, and induce a myosis from the unrestrained activity of the motor oculi nucleus.

In **tabetic forms** of general paralysis the oculi motor anomalies almost invariably *precede* the tabetic sign of abolished knee-jerk, and the pupils failing to dilate on shading or cutaneous stimulation, a **paralytic** myosis (moderate) eventually passes into a genuine **spastic** myosis (1 to 2·5 mm.), from the irritation of a disease process advancing upon the constrictor nucleus.

Cycloplegic Forms.—A special series of cases of general paralysis exhibit from early days a large-sized pupil, the site of an associative iridoplegia, which passes eventually into the **cycloplegic** form—*i.e.*, not only do the pupils fail to respond to light and on convergence, but paralysis of accommodation also prevails. Vision is unimpaired for distance, but *plus* glasses (+ 2 up to + 5 D) are required for close work; such ciliary paralysis is always bilateral. The pupils are affected not only through their **motor oculi** supply, but the sympathetic is also at fault, since no dilatation follows upon shading or sensory stimulation. Hence we have here the triple symptom of paralysis of the accommodative, the constrictor, and the dilator mechanism of the eye, and, as is indicated by Mr. Hutchinson, the ciliary ganglion is the only site, as far as exact anatomical knowledge extends, where the fibres for the tensor choroideæ, sphincter iridis, and sympathetic supply are closely associated, and could thus be injured by a single lesion.

It is, however, difficult to account for the slowly progressive nature of the paralysis, and the *constancy* of the bilateral implication, upon this assumption, and it at first appears rather to favour the view that the lesion is a slowly progressive degeneration affecting the several nuclei of the motor oculi, which, as clearly shown by Bruce and others, is very notably segmented. We regard it as probable that if, with the symptoms of cycloplegic iridoplegia, there is still response to sensory stimulation of the skin, but not to shading, the lesion is a nuclear degeneration of the third nerve; but if the reaction is abolished both for cutaneous stimulation and for shading, the lesion affects the ciliary ganglion. The remaining features characterising this group of progressive paralysis are :—(1) the absence of notable spinal symptoms; (2) the profound implication of speech ("drunken speech"); (3) the rapid course terminating in epileptiform convulsions and profound reductions; and (4) its frequent association with constitutional syphilis.

Spinal Symptoms.—In a large proportion of subjects of general paralysis the failure in the vigour and co-ordination of the greater musculatures comes on very gradually and insidiously; the lower extremities remain unaffected to any appreciable extent for even two or three years after the onset of the attack. Locomotion is unrestricted, equilibration is good, the gait steady, firm, and no swaying is induced on closing the eyes. In fact, in 50 per cent. of the cases examined the walk was brisk and not devoid of spring, and no muscular enfeeblement was apparent. Yet although the rule is that a gradually progressive paresis occurs, in a considerable number of cases a *sudden* paralytic seizure may occur, rendering the patient temporarily helpless in his limbs, or permanently paralysed with exalted reflexes and contractions established by consecutive spinal degenerative changes. In other cases, again, we find the deep reflexes *abolished*, and true tabetic symptoms obtrude themselves of *transient duration only;* on their disappearance, hemiplegia or convulsive seizures may occur, and symptoms of a descending lateral sclerosis come to the fore. The frequency with which we meet with spinal symptoms, and the general nature of these morbid signs, may be gleaned from an analysis of the forty-four cases before referred to. In six cases only (or 13·6 per cent.) were the deep reflexes ascertained to be perfectly normal; in sixteen cases (or 36 per cent.) they were decidedly exaggerated; whilst in some eleven cases (or 25 per cent.) the patellar reflexes were both abolished, or both very sluggish; in three more cases the knee-jerk was abolished on one side, and in a fourth very nearly absent. Thus we see a very notable degree of impairment of the deep reflexes characterises the affection, and the general results may be thus tabulated :—

DEEP REFLEXES.

Knee-jerk	normal on both sides	in 6 cases	(13·6 per cent.)	
,,	exaggerated	,, 16 ,,	(36·3 ,,)	
,,	absent	,, 7 ,,	(15·9 ,,)	15 or 34 per cent.
,,	very sluggish	,, 4 ,,	(9 ,,)	
,,	absent *on one side*	,, 3 ,,	(6·8 ,,)	
,,	nearly absent	,, 1 ,,	(2·2 ,,)	
,,	either exaggerated or sluggish	,, 4 ,,	(9 ,,)	
,,	doubtful	,, 3 ,,	(6·8 ,,)	

Increased Knee-jerk.—The exaggerated knee-jerk, it will be observed, is the more frequent phenomenon; it may be a purely functional disturbance, transient in duration, induced by nervous discharge from the cerebral cortex, and hence by removal of its inhibitory control. In this connection it is often found as the immediate result of a general convulsive seizure, or as actually accompanying the convulsive twitching of general paralysis; on the other hand, it may be a sign of organic disease of the spinal cord, and have as its accompaniments the usual motor enfeeblement and muscular contractions of descending sclerosis. The former association is illustrated by the table, which shows that out of twenty-two cases where the gait was elastic and brisk, seven present very notable increase of knee-jerk. It is important, therefore, to note that we have as associated phenomena in many cases of general paralysis, *a firm elastic walk, with full muscular vigour of limbs, exaggerated deep reflexes, and pronounced irido-motor paralysis.*[*]

The latter association—*i.e.*, of increased knee-jerk with structural disease of cord, is exemplified in cases *J. M.*, *J. B.*, *R. S.*, *B. K.*, and may be instructively associated with the ocular troubles as follows[†]:—

Case—J. M.—Right arm and both legs paralysed and contracted; knee-jerk notably exaggerated in both.; marked ocular paralysis.
,, J. B.—Right arm paralysed and rigid; right leg drags; knee-jerk notably exaggerated in right; marked ocular paralysis.
,, R. S.—Right hemiplegia with aphasia; right leg stiff; knee-jerk notably exaggerated in right; marked ocular paralysis.
,, B. K.—Left hemiplegia with contractures; knee-jerk notably exaggerated in right; horizontal nystagmus; ocular troubles.

Besides the above, several (three) of the cases of hemiplegia, with early commencing changes in the cord, were found associated with disturbed reflexes and marked intra-ocular paralysis.

Knee-jerk Abolished or Much Impaired.—We see by the table given above that fifteen cases, or 34 per cent., exhibited an abolition of the knee-jerk or its very notable impairment. Of the ten cases in which the knee-jerk is abolished, on one or both sides, five cases are notable for a brisk, elastic walk, yet all present serious oculo-motor paralysis; whilst of the remaining five, the gait is noted as being stiff and waddling; swaying, staggering, and leaning to right side; stiff, slow, swaying; and two others stiff. In all these latter the pupils are likewise fixed to their usual reflex stimuli. With one exception only, the deep reflexes when impaired, abolished, or intensified, were accompanied in all cases by irido-motor paralysis; but the latter condition was often found advanced with a perfectly normal reaction of the knee-jerk. Just, therefore, as we may find the association of an elastic, easy gait, or of a spastic or paretic gait, with exalted deep reflexes and advanced intra-ocular paralysis: so, on the other hand, we may encounter the association of a normal gait, or of a paretic or tabetic gait, with an abolition of the deep reflexes, and like irido-motor troubles. The **tabetic gait,** occasionally associated with this absence of the knee-jerk, is peculiarly disorderly, hurried, spasmodic, and insecure; the legs are jerked forwards, the feet planted wide apart, and the heels brought down with considerable force; there is often a tendency to propulsion; the patient sways from side to side or falls when the eyes are closed and feet approximated, or makes tottering efforts to secure his equilibrium. "Locomotor ataxy may be associated with other forms of insanity, and may in no great degree affect the duration of life; but in some other cases ataxy appears and is not complicated by mental disorder for several years, when other signs of nervous degeneration appear and general paralysis becomes manifest" (*Savage*).

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[*] As regards transient ankle-clonos in cases of general paralysis see p. 231.

[†] See "Degrees of Knee-jerk classified," by Leonard J. Kidd, *Lancet*, March, 1895, p. 801.

Bladder.—It is at this period that urinary troubles arise, and cause much anxiety to the guardians of the paralytic patient. When spinal symptoms have fully developed themselves and the lumbar cord is known to be involved, the patient is never secure from possible retention of urine, which, if not relieved by catheterism, may lead to a ruptured bladder; an accident frequent enough as to be a source of real anxiety, when large numbers of such paralytic cases are massed together in asylums.

In the earliest period of the disease, retention, or **incontinence,** may occur, but, as a rule, as a *transient condition* only; and at this time the patient is sufficiently conscious of his state to draw the medical attendant's notice to the point.

Retention may occur from spasmodic contraction of the *sphincter urethræ*, due to irritation of the lumbar cord, a loaded and torpid bowel being a most frequent starting-point for such troubles. It may be due, on the other hand, to the presence of a chronic cystitis and the alkaline urine so engendered, the cystitis having a neuropathic origin not infrequently in changes within the cord and spinal nerves.

Retention far more frequently is an indication of **paralysis** of the **bladder;** it is, then, usually accompanied by a dribbling away of water, which fails to relieve the gradually augmenting accumulation, and a time arrives when the organ becomes dangerously distended, and no expulsive power can be exerted by the patient. Such patients, by being constantly more or less wet in their bedding and clothing, would readily deceive an experienced nurse. The condition is identical with that induced upon section of the spinal cord above the level of the anterior and posterior roots of the third, fourth, and fifth sacral nerves—the sensory and motor arcs for the *sphincter urethræ*. Such section withdraws the inhibitory control of the cerebrum, thus increasing the reflex activity of the sphincter (*Landois**). All such cases should be uniformly treated by a periodic catheterism, allowing no great accumulation to occur.

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* *Op. cit.*, p. 653.

A still more dangerous condition arises in certain cases, fortunately somewhat rare. The bladder becomes *attenuated*, or undergoes considerable fatty degeneration, as the immediate result of spinal disease —a genuine tropho-neurosis. Nor is this very surprising when we learn from the results of autopsy in general paralysis how extensive are the trophic disturbances which other organs, and especially the *muscular*, undergo. In this degeneration the muscular coat of the bladder especially suffers, and the organ may be ruptured by a slight distending force when aided by such accidents as a fall or a blow, or even powerful expulsatory efforts, as in severe vomiting.

A recent convulsive seizure, or an apoplectiform attack, may leave the patient subject for some time subsequently to paralytic retention; and in our treatment of a case of this nature the state of the bladder should be almost the *first subject to engage our attention.*[*]

A similar condition of the bladder often prevails in advanced cases of tabes, and, as indicated by Dr. Buzzard, may even form the most prominent symptoms and, like gastric crisis, or optic atrophy, if ataxy be absent, be readily regarded apart from the real cause—" I have little doubt that not a few cases of atony of the bladder for which the surgeon is consulted are examples of tabes, with the bladder trouble predominating." [†] Apart from any well-marked spinal paralysis, retention frequently occurs as the result of simple inattention, the accompaniment of profound dementia, with which there is often associated a diminished reflex-excitability of the bladder, the organic reflexes corresponding to the general impairment of the *superficial* spinal reflexes. There is not only the diminished excitation of the spinal centre necessary to initiate the act, but the patient does not feel the *need* of micturition. It is found necessary in our asylum wards, where some sixty or seventy general paralytics are often congregated, to keep a daily and nightly record of all such inattentive cases, and of all bed-ridden cases alike.

Enuresis.—Incontinence of urine invariably occurs in the paralytic stage of this affection, as in all cases where the dementia also is advanced. It forms, together with like bowel troubles the daily source of trial to the nurse—a burden which may be considerably alleviated by tact and careful observance of simple rules of treatment.

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[*] A well-trained medical officer with many paralytic cases under his care will never fail to direct the nursing staff to keep a record of all such cases, and check the same himself by daily reference, morning and evening, to the warder's report, and by actual examination of the abdomen. Even under the strictest supervision an accident may still occur at times, as in the case of degenerated muscular wall of the bladder. It is, however, quite inexcusable for any such patient, known to be suffering from paralytic enuresis, to escape examination night and morning.

[†] " On little-recognised phases of Tabes Dorsalis " in *Diseases of the Nervous System.* Dr. Buzzard, 1882, p. 274.

Whilst retention is produced by section of the cord above the level of the reflex centres of the sphincter (above the third sacral nerve) by removal of its inhibitory centre, so section or disease on a level with the reflex centres produces incontinence, as will *any* incompetence in the reflex sensory or motor arc. It must be remembered that voluntary impulses passing down the motor tract of the cord do not act *directly* upon the smooth muscular fibre of the bladder, but they act in two directions—(*a*) on the sphincter urethræ or its motor centre in the cord, so intensifying the reflex contraction; (*b*) on an inhibitory centre in the cord above the reflex apparatus, which antagonises the latter and allows the sphincter to relax.*

The necessity for continuous care and change of bed-clothing in these wet cases is emphasised by the otherwise certain occurrence of bed-sore which, in these debilitated subjects, become a formidable complication to the nurses. The irritation of the skin, by its constant soakage in urine, develops, moreover, *papular eruptions* over the back, the groin, and thighs, which are abraded by the patient's hands.

Bowels.—Another troublesome and objectionable condition of the later stages of general paralysis, is the **paralysis** of the **anal sphincter,** which results in such frequent incontinence of the bowel; the condition, of course, is at once recognised on introducing the finger *per rectum,* when the patency and want of tone of the sphincter is very obvious. As is the case with the bladder and sphincter urethræ, the cerebrum can *voluntarily contract* the external sphincter ani, or can *inhibit its contraction;* such motor fibres descending through the cerebral peduncles to the lumbar cord.

The centre for this inhibitory agency is stated by Masius to be in the optic thalamus. So likewise, energetic voluntary contractions of the *levator ani* and *sphincter* arouse the active rectal peristalsis necessary to initiate defæcation, by bringing the excrementitious mass down into the rectum. When once there, it creates the uneasy feeling which prompts the voluntary inhibition of the *sphincter ani,* and allows the mass to be extruded. Thus the act of defæcation is in every way similar to that of micturition, it being really a **reflex spinal act** *during a* **voluntary inhibition** *of the sphincter.* There is the reflex loop constituted by the sensory nerves of the rectum, and the motor nerve of the sphincter and the *plexus myentericus* inducing peristalsis; a tract for voluntary impulse to excite contraction of the sphincter; a centre in the cerebrum for the inhibition of the latter.

Degenerative changes in the lumbar cord occasionally give rise to the complete paralysis of the anal sphincter; much more frequently is it a matter of sluggish or incomplete reflex of this muscle than of actual paralysis, as well as a defective tonicity which has been much alleviated by the application of tannin suppositories, a treatment first recommended by Dr. Robert Lawson.*

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* Landois and Stirling, *op. cit.*, p. 654.

In bed-ridden cases of general paralysis, a not unusual symptom is that of frequent alvine evacuation from simple increased peristalsis, not amounting to a genuine diarrhœa, but a very frequent "formed" stool; at times, however, the stools become very loose, yet without any pyrexial accompaniments, and due apparently to centric irritation of the *vagus*. Epileptiform seizures in general paralysis are apt to be accompanied or followed by such, but are then watery alvine fluxes. Thus in the case of *R. E. P.*, severe, continued convulsions, affecting the left side of the body only, were associated with very copious and frequent evacuations.

A similar condition has been noted by Dr. Buzzard in certain cases of tabes, and which he regards as possibly dependent upon irritation of the vagal nucleus in the medulla.†

In these cases the flux is probably the result of paralysis of the splanchnics, the vaso-motor nerves of the intestines; and to the resulting transudation of fluid from the blood-vessels into the bowel, with the accompanying increased peristalsis.

The Blood in General Paralysis.—A diminution of hæmoglobin is clearly indicated in all cases of general paralysis examined by us. The corpuscular richness varied considerably—in fact, from 75 to 126 per *hæmic unit*, the higher register pertaining to cases where maniacal excitement prevailed. No connection is established, however, between mania and such corpuscular richness, since a diminution in the number of red corpuscles is quite as often, and, in our experience, more frequently, met with in maniacal conditions. What is of more importance to note is the diminished colorimetric power of the corpuscle, the proportion of hæmoglobin varying from 52 to 75 per cent. Taking into consideration the corpuscular richness, we find that the absolute deficiency of hæmoglobin gives a corpuscular value varying between 56 and 89 per cent. The accompanying table gives the results obtained in fifteen cases of general paralysis at different periods of the disease:—

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* "Clinical Notes on Conditions incidental to Insanity," by Robert Lawson and W. Bevan Lewis. No. 1, *West Riding Asylum Reports*, vol. vi.

† *Ophthalmoplegia Externa with Tabes Dorsalis* (Dr. Buzzard, p. 200). See also case described by the same writer in *Diseases of Nervous System*, p. 218.

AMOUNT OF HÆMOGLOBIN IN THE BLOOD IN GENERAL PARALYSIS.

	Hæmoglobin.	Red Corpuscles.	White Corpuscles.	Value per Corpuscle.
	Per cent.	Per hæmic unit.	Per hæmic unit.	
T. G. (July 24, '87),	70	125	·40	·56
,, (Aug. 2, ,,),	72	126	·40	·57
W. W. (Aug. 5, ,,),	70	124·6	·13	·56
,, (Nov. 8, ,,),	70	108·8	·16	·65
T. C. (July 24, ,,),	60	103	·60	·58
,, (Aug. 3, ,,),	75	110	·50	·68
,, (Sept. 21, ,,),	63	85	·40	·74
W. A. (Dec. 16, ,,),	58	91·2	·20	·63
T. W. (Aug. 4, ,,),	52	80	·30	·65
,, (Sept. 29, ,,),	54	75·6	·40	·72
J. R. (Nov. 9, ,,),	66	102·4	·25	·64
S. S. (Oct. 9, ,,),	68	100·6	·24	·68
J. H. (Dec. 16, ,,),	60	86	·32	·69
J. B. S. (Dec. 16, ,,),	70	91	·22	·76
R. R. (Aug. 4), ,,),	62	81·8	·50	·76
,, (Sept. 29, ,,),	70	96·2	·32	·72
C. W. (Nov. 5, ,,),	64	79	·20	·81
J. W. (Dec. 16, ,,),	70	78·4	·22	·89
G. H. (July 17, ,,),	68	81·8	·20	·83
T. H. (Oct. 9, ,,),	...	77·2	·25	...
B. W. (Nov. 8, ,,),	64	..	·10	...

In the cases of *R. R.*, *J. B. S.*, *J. A.*, as of several others not noted in the above list, the blood flowed with great sluggishness, rendering its collection by the usual means extremely difficult. In such cases the surface was cold and very pallid, the vessels being undoubtedly in a state of spasm, and *instantaneous coagulation* was prone to occur, ere the blood could be withdrawn by the pipette; no inflammatory complication existed in these subjects. Similar cases of extremely slow oozing blood exhibited, on the other hand, abnormal delay in coagulation.

Clinical Groupings of General Paralysis.—We have elsewhere* endeavoured to analyse the more constant association of symptoms which characterise certain well-defined groups of this disease; and although we cannot here enter into a detailed account of such, we shall place before the reader a scheme of clinical groupings in which, as it appears to us, all forms of general paralysis may be comprised. Five clinical groups may thus be defined, according to the predominance of cerebral, bulbar, or spinal symptoms, the early or late onset of either, and the course pursued; they are as follows:—

Group 1.

Paralytic mydriasis; a partial reflex iridoplegia (light).
Increased myotatic irritability.
Excessive facial tremor and speech troubles.
Great optimism with profound dementia.

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* *Op. cit.*

Group 2.

Mydriasis with associated iridoplegia rapidly passing into the **cycloplegic form**—an early symptom.
Frequent myotatic excess, but no contractures.
Late speech troubles.
Acute excitement with frequent convulsions.
Very rapidly fatal course (preponderance of syphilitic history).

Group 3.

Spastic myosis ; a complete reflex iridoplegia.
Absent or greatly impaired knee-jerk.
Failure of equilibration ; locomotor ataxy, defective sensibility.
Very defective articulation.
Much optimism and excitement.

Group 4.

Late eye symptoms: paralytic mydriasis, a partial reflex iridoplegia (for light only).
Ataxic paraplegia confined to lower extremities (arms do not participate).
Great facial ataxy with extreme troubles of speech.
Epileptiform seizures ushering in pronounced mental enfeeblement.

Group 5.

No oculo-motor symptoms beyond occasional inequality.
No contractures, but notable myotatic excess.
No disturbance of equilibration, locomotion, or sensation.
Speech troubles not pronounced.
Epileptiform seizures very rare, *but from the first progressive deepening dementia.*

ALCOHOLIC INSANITY.

Contents.—Alcoholism and Age—Susceptibility at Certain Developmental Phases—Adolescent Period (F. S.)—Prevalence of Impulse—Influence of Sex, Heredity, Epilepsy, Cranial Injury, Ancestral Intemperance—Anomalies of Systemic and Visceral Sensation—Aural Hallucinations (J. J^1.)—Delusions of Suspicion—Optimistic Delusions—Clinical Forms of Alcoholism—Mania a Potu—Amblyopia Cutaneous Anæsthesias—Relapses—Case of W.W.—Homicidal Impulse (G.S.)—Chronic Alcoholism—Physiological Effects of Alcohol—Evolutionary Period—Mental, Sensorial, and Motorial Symptoms (J. J^1.)—Amnesic Forms (J. F)—Conditions of Mental Revivability—Delusional Forms (T. S.)—Instances of "Environmental Resistance"—Visceral Illusions—The Epigastric Voice—Various Illusory States (E. A. F.)—Evolution of Psychical Phenomena—The Nervous Discharge—Hallucination as Determining Morbid Ideation—Augmented Specific Resistance—Sensory Anomalies—Motor Enfeeblement (J. R.)—Twitchings, Tremors, Stolidity—Reaction-Time in Alcoholism—Muscular Spasms and Cramps—Oculo-motor Immunity—Nystagmus—Epileptiform Attacks—Hemiplegia (T. P. and J. C.)—Classification.

Alcohol is a fertile source of nervous disease, and its implication of the nervous centres is so general and far-reaching, that the resultant symptoms are of most protean nature; no poison, except the virus of syphilis, plays so extensive a *rôle* in the morbid affections and degenerations of the tissues, nervous or non-nervous. Yet, as regards its effects upon the nervous system, it is possible to trace its march with a fair degree of accuracy, and to classify into definite groups the victims of over-indulgence in accordance with the degree of implication—the depth to which nervous dissolutions have attained. Ere we classify, however, the more or less distinctive forms of such affections, it will be well to glance generally at the insanity induced by alcoholic indulgence; and for this purpose we have inquired into the history and antecedents of 464 patients, whose insanity was attributable to excessive drinking; of which number 344 were males. And, in the first place, who are the subjects most liable to the different forms of alcoholic neurosis?

Age.—The period of life is here an element which it is important to examine. Were we acquainted with the actual amount of excessive drinking in the community at large, and at different ages, as also with the percentage of those who succumbed to insanity as the direct result of drink, and the time required for excessive drinking to evolve such results, we might, by a comparison of asylum statistics, ensure some degree of accuracy in estimating the incidence of alcohol as a causative agency of insanity. Such absolute data are at present out of our reach; and we must, consequently, rest content with the ascertained history of our *asylum community* without reference to the *sane.* Nor is this altogether devoid of immediate utility, since our object is not so much that of ascertaining the exact incidence of alcohol in insanity, as to extract the characteristic features of the neurosis which alcohol induces. Every period of life shows its proclivities towards special disease; and the action of toxic agencies demonstrates the peculiar susceptibility of the nervous system to their operation at certain stages of its evolution. Some such law would appear to govern the origin of mental affections induced by alcoholic indulgence, since these are certainly far more prone to occur between the ages of twenty-five and thirty, and, again, from thirty-five to forty-five, than at other periods of life. It is easy to assume that at these periods of life the actual number of excessive drinkers is larger than at other times; at present no data supporting such assumption are forthcoming, nor do we see any reason why the age of thirty to thirty-five should claim special immunity. It must be remembered that this age, from twenty-five to thirty, is one peculiarly characterised by intellectual advance, as contrasted with the more emotional developments and expansion of the moral nature which takes place during adolescence.

It is also the age when the struggle for existence, in its widest sense, makes itself felt upon the organism in fullest force; it is not the period of longing and yearning for activity, for *plans* of action and castle-building, but it is peculiarly the age of *active being,* when the *mettle* of the man is tried, and his weight as a social unit fairly estimated. It is upon his intellectual advance, which at this epoch is so important and so notable, that his success as a social factor largely depends; for a successful life is the outcome nowadays of a well-balanced adjustment, and hence depends on a highly appreciative and intelligent recognition of complicated relationships.

It is a period when feeble and indifferent organisations often feel a want for an artificial stimulus to goad them on, and many succumb to such perilous inducements; and it is peculiarly a period when certain inherited neuroses place the individual at a disadvantage in the competition of life. In fact, it is a period when the first great swellings of the intellectual tide make themselves felt throughout the whole organism, and when inherited frailties, coeval in their manifestation in parent and offspring, assert the supremacy of the laws of periodicity in development. All such nascent developments are most prone to early decay in dissolutions of the nervous system; and upon them chiefly appears to be expended the full force of those agencies credited with the proximate causation of insanity. Thus it is that in the *moral and emotional developments* of the adolescent epoch, sexual and alcoholic excesses tell more directly upon this phase of mental life, and that hysteric forms of insanity and a stunted moral development are so often revealed at such an age. In like manner, this latter epoch of intellectual expansion exhibits the earliest effects of alcoholic excess as inducing reductions in the intellectual sphere, and only later on, as profoundly affecting the emotional and moral being of the individual. This is why we regard age as an important element in the evolution of these forms of alcoholic insanity.

F. S., aged twenty-five, widower, and a warehouseman. When admitted he had been insane for six weeks; had been very wild, rambling in speech; called himself the "Holy One," the "Great Physician." Patient's father is of dull intellect and of intemperate habits; paternal uncle was insane; patient was addicted to excessive drinking from the age of fourteen to that of twenty-one, remained temperate for two years subsequently, and has again relapsed into his former excesses. Upon admission he exhibited great exaltation, spoke excitedly, and loudly, giving expression to optimistic delusions; he had exalted notions respecting his muscular powers; was "perfect in body and mind, and surpassed all others in knowledge and skill;" he has "a perfect knowledge of the human frame, is a great physician, and can cure all diseases." He declares that he can "easily lift half-a ton, and has often raised many hundred tons aloft; all England will become his, ere long; is possessed of enormous wealth." His manner is abrupt, but he is inclined to be friendly and jovial; expression flushed and excited; pupils widely dilated, but equal and of normal reaction; tongue shows notable and extensive fine fibrillar tremor, no ataxic jerks; articulation is unimpaired; the reflexes are normal; cutaneous sensibility is unimpaired. Patient is muscular and well nourished. Examination of other systems proved negative.

In a fortnight he was considerably calmer; the same *bien-être* was manifest, but he was so far reasonable as to be employed. This remission lasted but two weeks, and he relapsed into severe maniacal excitement, in which with every varying mood, from abrupt rudeness to jovial humour, he maintained the same exalted, grandiose notions. His habits now became degraded and filthy at night, and masturbation was practised.

Six months after admission, excitement continued unabated, he was insolent, threatening, and demonstrative. Habits of masturbation so repulsively shameless and open that the liquor epispasticus was applied locally, and chloral with bromide of potassium given internally with only temporarily good results. These habits kept up persistently seemed to account for the slow progress made in his case, for he remained twelve months in the asylum ere the excitement abated; even then for several months he exhibited an imbecile aspect, laughed immoderately without cause, was restless, untidy, senseless or irrelevant in his observations, and given also to insane gesticulation and grimace.

Twenty months elapsed ere he was discharged recovered.

We must, as before hinted, make due allowance for this age as one offering peculiar inducements to heavy drinking; and for the fact that a certain period, even for those specially predisposed, must elapse ere alcoholic excess results in actual mental alienation; but, when all such factors are allowed for, we still think the evolutional phase of this epoch is the chief reason why so large a proportion of mental cases are attributable to alcoholic excess.

The facts as given in our statistical Tables are striking, for out of 344 males suffering from one or other of the forms of alcoholic insanity, 29 cases alone occur between the ages of fifteen and twenty-five, whilst as many as 52 occur during the next five years, or 87 up to thirty-five years of age; each of the two succeeding quinquennial periods of life claiming some 50 victims of these affections.

Predisposition.—The subjects of alcoholic insanity admitted into our asylum do not exhibit any unusual degree of the *insane* heritage, the proportion of hereditary cases not rising above 27 per cent., and, consequently, not attaining to the average heredity of *all forms of insanity alike.* All recurrent cases of insanity taken together exhibit a far higher insane inheritance than this. If we now group together all cases of insanity, epilepsy, and other neuroses, occurring in the family history of these insane subjects, as also all cases of ancestral intemperance, we find such predisposing elements present in 37·2 per cent. of the total number of cases of male patients. Where ancestral intemperance was the sole ascertained predisposing cause, it was almost exclusively limited to the father, and in no case was the mother addicted to this vice. Taking a history of insanity and excessive drinking collectively, we find such present in the case of thirty-one fathers and sixteen mothers, so that the influence of sexual limitation in transmission is here apparently demonstrated.*

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* See in connection with the question of alcoholism as a factor in the production of epilepsy, an elaborate and instructive article, "Heredity and Crime in Epileptic Criminals," by Henry Clarke, *Brain*, vol. ii., p. 491.

Nature of the Attack.—Taking first the 344 males—maniacal excitement prevailed in 57·8 per cent., of which over 26 per cent. are delusional forms of insanity, only 6·3 per cent. being acute maniacal states. On the other hand, melancholic depression prevailed in 28·7 per cent.; 42 cases were attended with delusional perversion, 28 were simple melancholic forms, while 12 (or 3·4 per cent.) were cases of chronic cerebral atrophy. The maniacal states were, therefore, considerably in excess of the melancholic forms of alienation, in fact, they were twice as numerous; whilst pronounced dementia appertained to a small section, forming only 8·4 per cent. of the whole.

Taking the aggregate of 344 cases where alcoholic excess preceded the attack of insanity, the first important fact taught us by a glance over our statistics is the essentially *impulsive nature* of the affection; it is in all its phases a **convulsive neurosis.** Whether excitement prevails, and the disordered propensities exhibit sudden, explosive impulses; or whether depression, with its frequent accompaniment of hallucination, predominates, and painfully pent-up feeling, or suddenly-aroused terror results in determined violence to self or others; or, lastly, whether they are forms of mental fatuity with depression—the all-important feature to be borne in mind is this prevailing convulsion of conduct. The maniacal forms exhibit such impulsiveness, not so much in attempts at self-injury as in a **dangerous aggressiveness** to others, in destructive fits, in sudden, treacherous, and often brutal violence, a tendency which renders these lunatics a peculiarly dangerous element in our asylum communities; about 68 per cent. were thus returned as dangerously impulsive towards others. The melancholic victim, however, is more likely to turn his hand against himself; one half of such cases at the lowest estimate being **dangerously suicidal.** The tendency to suicidal and homicidal impulse is high even in advanced forms of dementia, and it is a noteworthy feature that in those cases of dementia which are dependent upon chronic alcoholic cerebral atrophy, suicidal and homicidal impulse reaches its climax of frequency; as many as 66·6 per cent. of such forms being determinedly suicidal, and 83·3 per cent. being dangerously aggressive. The intrinsically impulsive outbursts of alcoholic insanity, whether mania, melancholia, or dementia prevail, should never be forgotten by those dealing with the insane.

Taking into account only the male alcoholics, **age** apparently had no distinct influence over the *character* of the mental symptoms, one half the cases of mania, as of melancholia, occurring up to forty years of age, and the other half, subsequently. We may anticipate the largest number of maniacal or melancholic patients to be between twenty-five and thirty years of age, and the next largest proportion to be in the quinquennial periods immediately preceding and following the age of forty. A considerable rise in the number of melancholic cases amongst such a class of insane inebriates again occurs at the age of fifty to fifty-five, and a similar rise in maniacal ailments from fifty-five to sixty years of age. We may, therefore, conclude that although certain periods of life are especially prone to the development of alcoholic insanity, such as the ages of twenty-five to thirty, from thirty-five to forty, and again towards forty-five, maniacal and melancholic forms appear in the same relative frequency at these epochs of life.

If we attempt to explain why the form of insanity should assume in one case the maniacal and in the other the melancholic type, we are able to afford but little explanation and that purely of a negative character. Thus age is, as just noted, an indifferent element in this connection; in like manner **inheritance** cannot be stated to have any very definite influence in either direction; excitement does, however, predominate in hereditary insanity; but the proportionate number of maniacal to depressed cases appears still greater among those who afford the history of *ancestral intemperance.* **Epilepsy** and **other neuroses** also appear to be wholly indifferent factors. Then again, as regards **sex,** it is noted that melancholic states are to maniacal proportionately more frequent in male than in female inebriates, being but one-fourth in women and one-half in men. Sex, therefore, does appear to lend some influence in predisposition to the one or the other type of insanity. Lastly, **recurrent seizures** throw no light upon the subject, depression and excitement occurring with about the same relative frequency in relapsed cases (mania, forty-three, and melancholia, thirty-seven). **Cranial injuries** occur in a large proportion of the subjects of alcoholic insanity (18·9 per cent.), but this element comes in as frequently in maniacal as in melancholic states. Of the circumstances which modify the type of the psychosis age, recurrence, and cranial injury may be excluded from consideration; whilst sex, heredity, and ancestral intemperance have some influence in this direction.

Hallucinations of Special Senses.—Illusions and hallucinations are extremely frequent in all the acute forms, as well as in a large proportion of the chronic forms of alcoholic insanity; in 344 males as many as 131 (or 38 per cent.) presented such disturbed sensorial phenomena. The visual were the more frequent, and visual or aural were separately more frequent than both combined. But what is peculiarly characteristic of these alcoholic forms of alienation are the illusory and hallucinatory phenomena of the nerves of **general sensation** and of the **systemic** or **visceral system** of nerves, giving origin to delusions of an extraordinary nature, and often of a very complicated system of intrigue. Tingling, prickling, burning, stinging sensations over different areas of the integument are frequently complained of; anæsthetic patches are discovered over the skin of the arms and face, and a feeling of general numbness in a limb may ensue; electric-like shocks are described in the limbs, and head, and neck, often associated with muscular twitching, or facial contortions; and these subjective states, induced usually by centric changes, are referred to an objective origin, giving rise to the most varied delusional concepts, such as those of unseen, mysterious agencies operating upon the system—electricity, magnetism, mesmerism, witchcraft, diabolical machinery are in turn invoked to account for these mysterious sensations. In like manner, unusual visceral sensations referred to the heart, lungs, stomach, bowels, &c., become the basis for similar delusional beliefs of a malign influence within. Belief in demoniacal possession is not uncommon, but more frequently is the imagined torture supposed to be produced by individuals known to the patient, who, he believes, have the power of operating upon him from a distance, or have obtained access to his body, and restrict, enslave, and govern the whole life of his organism, control his thoughts, and have dominion over his mind and its utterances.

J. J^1., aged thirty-one; admitted March, 1886. Had been a soldier, and for the past five years on service in India; he was invalided by "fever," confined to a military hospital, and then sent home to England. During his voyage home, a "galvanic battery began to play upon him," and he heard the voices of his late officers, Capt. P., Lieut. C., Drs. W. and C., talking of murders and other crimes, although they were not present. He has heard these voices persistently since coming to the asylum; they are always above him, and he points up to a distant roof of the building where he believes they are located. He often hears the whistling of gas over his head, which, he says, affects him so as "to snip a word in two," just as he utters it, and confounds the meaning of what he says—it also affects his memory; this gas is produced by the same agencies as the voices which he hears. Flashes of lightning show him all the events of his life. "I have seen my whole life, good and bad, in yon back-yard" (referring to an airing-court). The battery sends electric shocks through his body, causes a heavy pressure (not a pain) at his epigastrium, twitches up his chest, but does not affect arms or hands. His speech is hesitating, and he often, in explanation, uses the statement that, "*They* rule my speech, and tell me what I have to say at times." Has noticed foul odours, which he knew were unnatural, and caused by "the electric machine;" they prevent him from sleeping. These malevolent agents are treacherously pursuing him wherever he goes; he knows not why—he cannot rid himself of them, although he has "offered them his life." Frequent twitchings of the facial muscles on the left side occur, and he explains them as due to the electric shocks, which draw his breath out of him at these times; his "head shakes," and his eyes "are made to twitch thereby." He admits having been of very intemperate habits since the age of eighteen, but had never suffered from *delirium tremens*; both his father and mother were excessive drinkers. He himself drank raw spirits freely. Had never suffered from fit or stroke.

Dynamometer registers for right hand 56 kilos.; for the left hand 54—as the average of four trials.

Æsthesiometer gives the following measurements of comparative sensibility :—

	RIGHT SIDE.	LEFT SIDE.
Tip of forefinger, . . .	·05 of an inch.	·05 of an inch.
,, thumb, ...	·05 ,,	·10 ,,
Ball of thumb, ...	·3 ,,	·4 ,,
Centre of palm, ...	·4 ,,	·4 ,,
Wrist, dorsal, ...	·9 ,,	1·3 ,,
Wrist, volar, ...	1·7 ,,	1·4 ,,
Forearm, dorsal, . . .	2·3 ,,	1·7 ,,
Forearm, volar, ...	1·9 ,,	1·8 ,,

Sensibility elsewhere appears good, active, without delay; yet he complains that his legs frequently feel "dead," as he sits at table. Both knee-jerks are quite abolished ; yet equilibration is undisturbed, he balances well with eyes closed ; stands on tiptoe, and can walk "heel and toe" along a straight line ; plantar reflexes are good. Has never had pains in his limbs, but flashing pains continually pass through his body in "all directions." No eye-symptoms are apparent, the pupils are equal, the reflexes perfect ; has never suffered from diplopia or strabismus.

On analysing the varied delusions in male alcoholics, which were well expressed in 208 out of 344 individuals, it was found that 131 entertained ideas of persecution; 29 others, religious delusions affecting their moral welfare ; and the remaining 48, optimistic and grandiose conceptions ; or, as tabulated, thus :—

NATURE OF DELUSIONS IN ALCOHOLIC MALES.

	Cases.	Percentage of whole (omitting decimals).
Delusions of Persecutions—		
(*a*) By poisoning, ...	24	12
(*b*) By magnetic and unseen, mysterious, agencies,	25	12
(*c*) By various other means beyond the above, .	82	39
Delusions affecting the moral being, ...	29	14
Delusions of grandeur and of wealth, ...	48	23
	208	100

Frequency of Delusions of Suspicion.—Thus, about 63 per cent. of such false notions are of the nature of *delusions of suspicion*, and of the 23 per cent. of a grandiose and optimistic character, it was also observed that such notions were very rarely unmixed with distrust and suspicion—the exalted position—the large possessions or wealth of the individual being cited as in themselves the explanation of the malignity of his imaginary foes.

A summary of all the cases of delusions of mysterious or unseen agencies, based on illusory states of general or visceral sensibility, vividly suggests the terrible mental torture which these alcoholic subjects endure. It should be remembered that the prevalence of these latter forms of delusion, based on illusions of the nerves of visceral and general sensation, is much greater than our statistics would lead us to infer, since therein are comprised only *definitely expressed* states of the kind, while a much larger section exhibit suspicious evidence of these.

Optimistic Delusions.—These states of optimism closely resemble those presented by the subject of general paralysis, in the *intensity* of the false belief, and their grossly exaggerated character, but they differ in almost invariably exhibiting the feeling of **distrust** just alluded to, and their **far greater fixity.** The subject is restrained in the exercise of his exalted mission, or in the recovery of his just rights; his functions, delegated by the Almighty or by a great earthly potentate, are checked by the malignity of his former friends and relatives, perhaps by his own wife and children, to all of whose actions sinister motives are attributed. Aural hallucinations prompt him to action—a voice from the heavens declares to him his mission—yet his enemies thwart him, endeavour to poison him, or otherwise ill-treat him, and this leads to frequent impulsive violence. Yet, when contrasted with the other forms of delusion of persecution, it is found that hallucinations which are found in one-half of these cases are not so frequent an accompaniment of the exalted mental states, occurring in but one-fourth of the series. The general character of these delusions may be gleaned from a few typical cases—thus one of our patients calls himself the "Son of God, and the Father of all nations;" another declares he holds the sun and moon in his hands, and regulates the movements of the planets; another has been left a fortune of one million pounds sterling by Baron Rothschild; another has just produced a great patent whereby his fortune is secured. One acute case (recovering in the course of four months) declares that he drives six of the finest horses in the world. Noble ancestry is boasted of by some, or matrimonial alliance claimed with members of royal blood; and one of our most acute cases always spoke of his wife as Queen Elizabeth, and was possessed of fabulous wealth; the son of another was so wealthy that he was about to buy up Wakefield.

Delusions of persecution comprise, as we have before stated, nearly 63 per cent. of the whole series, with the very frequent association of hallucinations of the special and general senses. A very large proportion of such entertain ideas of poisoning—their food, medicine, or tobacco is drugged; attempts are made to stupefy them by chloroform, to smother them when asleep in bed, and to burn them alive; ideas of murder in every conceivable way are rife; their house is to be blown up; they are to be "cut in pieces and boiled," or divided limb from limb, and "their buried children disentombed." Policemen dog their footsteps; soldiers lie concealed in their houses; voices are heard next door intriguing with the wife against their life; rats and vermin surround the bed; the wife's fidelity is frequently called in question. These are some of the more prominent instances occurring in our series of male alcoholics, of which details are afforded in the Table.*

Of the Clinical forms of Alcoholic Insanity.—We shall now proceed to a study of the varied forms of alcoholic insanity, under their respective headings of acute and of chronic alcoholism; premising, that by the former we indicate a purely toxic form of insanity in which the mental derangement (often very acute as regards intensity) is of rapid course and short duration—a more purely *functional* derangement, due to the presence of the poison in the system; and that by the latter we refer to the more remote effects of the poison in altering structure, through modifying the nutrition of the cerebro-spinal system—an insanity based upon *organic disease* of the brain and spinal cord. The statistics already dealt with when considering alcoholic insanities generally, have presented us with some 50 per cent. of cases running a rapid course towards complete recovery; but in which there are also some 40 per cent. of others whose recoveries were very partial, or death resulted, or the patient remained an addition to the chronic insane community. It is upon such categories we shall now draw for illustrations of the various phases presented by the mental perversions induced by prolonged alcoholic excess.

Acute Alcoholic Insanity.—Under this term we comprise *mania a potu*, or the acute alcoholic delirium of Magnan, and *delirium tremens*, or "febrile" delirium tremens of Magnan.

Mania a potu (acute alcoholic delirium; delirium ebriosum).—Our patient usually comes before us in a state of acute maniacal excitement, and with some such history as the following:—He has been for a long period addicted to intemperate habits—perhaps, not so much *continuous, heavy drinking*, as repeated excesses, often with prolonged intervals of comparative sobriety between the bouts. There is probably a clue to one or more attacks of delirium tremens, from which on recovery he has shortly relapsed into his former excesses leading to an acutely-delirious outburst.

It is by no means unusual to be told that, for several weeks prior to the seizure, there had been entire abstinence from alcoholic indulgence; but that the health had been notably affected, with gastric disturbance and general malaise; nervous symptoms had been prominent, and mental instability, moroseness, irritability, insomnia, hideous dreams, and nervous startings had been witnessed; and that, consequently, on the occurrence of some moral agency, shock, grief, disappointment, &c., an exciting cause is afforded sufficiently potent to develop the attack of mania. Our enquiries probably elicit the fact of hereditary predisposition to insanity—possibly of ancestral intemperance; but especially are we likely to discover that the subjects have been regarded as congenitally defective in self-control, as wanting in moral tone, and as the victims of a stunted development, in which instinctive desires and impulsive responses predominate over higher intellectual promptings. The excitement is often one of great intensity; but, in this respect, we witness various depths of reduction, yet all forms are invariably accompanied by characteristic illusions and hallucinations; in fact, the most notable feature of the delirium is the predominance presented by such sensorial disturbance. In typical *delirium tremens* motor symptoms are as prominent a feature as the sensorial; whilst in the more chronic forms of alcoholism, as we shall see later on, we get both features less emphasised, less acute, and, together with intellectual enfeeblement, assuming a permanence wanting to the acute forms.

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* For a clinical study of the persecutory delusions prevalent in various forms of insanity, see "Insanity of Persecution," by Réné Semelaigne, *Journ. Mental Sc.*, vol. xl., p. 500.

The special sense illusions and hallucinations are ever of a most distressing nature, usually very vivid, and exhibit the usual mobile state of such sensorial anomalies seen in acute mania. This fleeting character is in itself of favourable augury when contrasted with the more persistent fixity, or monotonous repetition, seen in other states of mental disease, and indicative of an approaching or of an established chronicity. The variable, fleeting nature of the sense-disturbances in alcoholics has been long recognised (*Lasègue*, *Magnan*). The forms thus conjured-up by the disordered sensorium bear a striking resemblance to the other form of acute alcoholism, *delirium tremens*, as also to the phenomena described as induced by certain drugs, notably hyoscyamine (*Robert Lawson*). As under the influence of hyoscyamine, pleasurable or painful visions troop before the mind's eye *incessantly;* yet the general mood in acute alcoholism is always painful, and the visions, however fascinating in character, beget distrust and suspicion. Much more frequently are these false impressions of a most painful, terrifying nature; and hideous, loathsome forms surround the victim, Snakes, tigers, furious dogs are seen or heard, and the attendant is transformed by the diseased mind into a fiend or other dreaded form.

If we now test our patients carefully, we discover in many a very decided degree of *amblyopia*—vision is clouded, and the visual activity diminished; and, with the amblyopia, there is also occasionally conjoined a difficult perception of colours (*dyschromatopsia*). It has been shown by M. Galezowski that the chromatic anæsthesia thus produced pertains chiefly to the composite colours, and especially yellowish- and bluish-greens. Impaired or perverted sensibility may also be recognised in other sensory expansions, as the olfactory and gustatory;

the palate is in all cases more or less affected, and the anæsthetic condition of the upper lip is an early symptom familiar to all who indulge too freely in alcoholic drinks. Similar anæsthesias, hyperæsthesias, and perverted states of general cutaneous sensibility have likewise been appealed to as explanatory of the many forms of illusion pertaining to the surface of the body from which alcoholics suffer. That these sensorial expansions do become affected seriously in acute alcoholism is undoubted; but such symptoms are of transient duration, and are far more frequent in chronic alcoholics; they but indicate the taking off "of the fine edge," which all mental faculties alike suffer from as the result of alcoholic reductions.

Relapses.—Alcoholic excess, long ere structural change can be predicted in the nervous centres, is answerable for something more than the mere transient functional disturbance described; it engenders a nutritive perversion, which is more marked after each attack of acute alcoholism, and which is expressed in a notable tendency to recurrence. This relapsing character is especially seen during the progress of the alcoholic subject under treatment; repeated outbursts of excitement occur, after intervals of comparative calm and often apparent convalescence, ere the case may be considered fit for discharge from asylum-supervision. Thus, in the case of *J. J.*, four distinct relapses occur during one year of his residence at the asylum, and although the remissions were not so complete as in many cases, yet it was sufficiently apparent in his case that each relapse was characterised by symptoms exactly reproducing his previous state; and that the immediate exciting cause was some trifling moral agency, such as a dispute with a patient, or some trivial disappointment. It is all-important for us to recognise the fact, that the *presence* of alcohol in the blood or tissues is *not* necessary to the continuance of the characteristic delusions of persecution, to which these individuals are subject; it is in the nutritive change engendered in the nerve-cells of the cortex through the agency of alcohol, that a more permanent instability of the discharging centres becomes established, and the mental anomalies assume gradually a more stereotyped aspect. Whatever be the centres of the brain which are more prone to disturbance through the agency of alcohol—when once their nutritive equilibrium is upset seriously by this agency—these centres are *prone to suffer first* in any relapse, whatever be the exciting cause.

The case of *W. W.* will illustrate this point:—

W. W., aged forty, coal-miner; admitted February, 1885. Mother had been an inmate of this asylum, and was said to have died in Pontefract Hospital from softening of the brain. The patient was a heavy drinker until ten months previous to his entry into this asylum; an attack of mental disorder, the nature of which is unknown, but which was treated at home, served, however, to check his habits of intemperance. From that time he worked steadily, at such scanty employment as he could procure, till within a week of his admission here, when he was seized suddenly with symptoms of excitement and ravings on religious topics; this speedy onset was attributed by his friends, to his attendance at the Salvation Army meetings and consequent excitement. On his reception into the asylum, he was suffering acute mental depression, and was too agonised to offer any information regarding his subjective state; but, according to the certificate, he had avowed the delusions that "there were devils inside him, and that a man had come outside his house to attack him," and he had taken up a poker in order to kill him. In a few days, having quieted down, he affirmed that he heard people coming down on the top of his head, and although he could not remember what they said, comprehended it at the time; was fearful of sleeping at night. Rapid convalescence supervened, delusions and hallucinations disappeared, and the patient was discharged six weeks after entry.

Here then, we find, after nine months' abstinence, the recurrence of acute melancholia apparently attributable to the morbid excitement of certain religious services. In every feature the attack reproduced what was previously engendered as the direct result of heavy alcoholic indulgence; and it is well to be familiar with the fact, that the symptoms of acute alcoholism may thus be over and over again reproduced, without fresh excesses, when the cerebral nutrition has been impaired as above described. It is noticeable how, in the case just described, the characteristic hallucinations and delusions were also freely interspersed with religious delusions, and how his ramblings brought prominently into relief the subject with which he had been chiefly occupied at the onset of his attack. As Magnan and others have noted in other cases, here also the hallucinations gradually lose their definiteness, a confused voice replacing the alarming cry of "poison!"; then the voices are in their turn replaced by an occasional humming sound in the ears, which ultimately fades away upon his recovery. It is impossible not to be impressed, when attentively studying such gradual recoveries, with the apparent obnubilation of the illusory states by the *strengthening impressions of objective existences*, forcibly reminding one of what occurs occasionally, even in perfectly healthy states, when awaking from sleep; illusory states are then not infrequent ere more vivid presentative feelings force themselves into being. In a case of *mania a potu*[*] (*W.R.*) special interest attached itself to the visual illusions to which the patient was subject, especially at the moment of waking. It was, as it were, a projection of a dream into his waking hours, fragments of the illusory dream persisting and refusing for some little time to be dispersed upon the re-instatement of wakeful consciousness. This state is not unfamiliar in normal health; and a case is known to the writer, where for some time after apparently complete wakefulness, the subject saw distinctly what he conceived to be his own corpse lying in a coffin beside his bed, and which for some time he failed to resolve into its real elements of a bundle of clothing. We reasonably conclude that such resolution is affected by the freer circulation in higher cortical realms; and that zones previously anæmic become, on complete wakefulness, once more the site of functional activity. There is a strong presumption that a parallel condition exists in acute alcoholism, and that a projection of hideous dreams and fragmentary detached illusory states are thus intermingled with the realities of waking hours; the whole history of the case during its acute stage is that of a waking dream. The re-energising of higher cortical planes which occurs during waking may require a certain well-defined interval, and in lieu of *dispersing* any existent morbid symptoms will, in certain conditions, call them into full activity as in the movements of paralysis agitans. Thus in a case of Charcot's hemiplegic type of *paralysis agitans*, the writer well recalls the statement of the patient that the hand which was the site of continuous fumbling movements during complete consciousness, and especially during voluntary action, remained often quiescent for some time after waking—a very appreciable interval existing before the affected centres were sufficiently energised to permit of their intermittent discharge.

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[*] See on this point, *Magnan*—Transl. by Dr. Greenfield, p. 50.

In the case of *W. R.* it is also to be noted that both he and his grandmother "could foresee events," by which we may infer that both were subject to these peculiar waking dreams, and were apt at such moments to confuse illusory appearance with actual existence, and visions arose before them in their waking hours. It is by no means unusual amongst the insane to discover a power of calling into existence such illusory appearances; and we are frequently told by them that they have the power of conjuring up almost any form they choose; nor is this to be wondered at, if the analogy of dreaming be considered; for we opine that the morbid imagery is always ready (in certain cases) to spring into life, but is suppressed by the attentive direction of the mind to presentative states; if, on the other hand, such contrasting states are voluntarily suppressed, the morbid imagination may have full play. At all times liable to dangerous impulsiveness, the acute alcoholic is *a fortiori* more prone to exhibit such impulses *at night*; and especially, when roused from slumber, at the *moment of waking*, from the occurrence then of vivid, illusory, and hallucinatory states. A colleague of the writer's thus narrowly escaped with his life a violent attack on the part of a patient, who had concealed beneath his bedding an improvised weapon, with which to attack the medical officer at the night-visit to his bedside; and who confessed subsequently that *each night* he had imagined his visitant to be under the form of Satan, and planned this means of attack upon him. Such impulsiveness very frequently betrays itself in suicidal attempts; and we find by our statistics as many as 40 per cent. regarded as decidedly suicidal. According to Bouclereau and Magnan, from 7 to 15 per cent. of alcoholic cases attempt suicide. The latter writer is especially guarded in distinguishing genuine suicidal and homicidal attempts from mere accidents, which are, of course, peculiarly prone to occur in the terror infused by the delusional states of acute alcoholics. Such suicidal impulses may be associated with desperate conduct, not truly homicidal nor suicidal, but having as its object the relief of the existing torture.

G. S., aged forty-seven, married, a woollen spinner by occupation. For two months prior to admission he had been depressed, sleepless, and had taken but little food. A fortnight before he would not leave his house, was silent, sullen, and obstinate, betraying much terror because he was "to be taken away and deserved hanging." Wife stated that for years he had been an excessively sottish drinker, but less intemperate for the past six months. Brother was insane. Patient was a fairly nourished, muscular individual, well built, with a heavy, stupid expression, sluggish in all his movements, his whole bearing indicative of great apathy. He was very illiterate; was reticent, wilful, and refused food upon admission. There was no oculo-motor paralysis; tongue was protruded straight and steadily—it was covered with foul epithelia; heart's action feeble, no murmur. Abdominal viscera apparently free from all but slight functional derangement.

During his first week's residence, when sleeping under observation, he suddenly sprang out of bed, threw himself upon a patient next to him without any provocation, and nearly strangled him; he was removed to a single room where he was discovered mutilating himself, having succeeded in inflicting a deep incision with his finger nails around the penis.

Up to this period he had been taking morphia; hyoscyamine (⅛ gr. Merck's Extract) was now ordered. A month after admission it is noted—"Much quieter, but still has a hang-dog look, as if much afraid of something or somebody;" and, a few days later, he became greatly excited and suspicious, attacking his night-attendants and fellow-patients. Chloral (grs. xxx) ordered night and morning.

Muscular enfeeblement, *especially of the lower limbs*, was now noted; in his wild excitement he frequently fell and bruised himself badly, so that he had to be confined to his bed in a padded room. Six weeks after admission, the excitement had passed away; patient was left extremely depressed in spirits and *profoundly demented*; was very restless, and utterly negligent in habits.

He had at this time the aspect of an advanced general paralytic, but with no labial, lingual, or ocular paralysis. Some paralysis of the muscles of deglutition subsequently supervened, necessitating very cautious feeding. He remained helpless, bedridden, and extremely demented, dying somewhat suddenly six months after admission.

The case of *J. B.* (p. 249), is a typical one of *mania a potu* passing into chronic alcoholism in a subject predisposed to insanity, and inheriting the results of paternal intemperance. Prior to his visit to America, his seizures were of the nature of acute alcoholism; but, upon his return to England, the fixed delusion of the machinery in his chest augured the transition towards chronic alcoholism. In the latter stage we observe the tendency to allude to his sufferings as terrible, and to speak in the most exaggerated terms of the tortures to which he is subject. This is a feature highly characteristic of chronic alcoholism; such exaggerated statements are not wilful misrepresentations, for the subject fully conceives the terrors he depicts. The suffering is evidently not extreme physical suffering, but a distortion of disordered sensations, so that slight pains and discomfort, from a loss of balance in comparison, are apt to be magnified into voluminous distressing feelings. Such subjects usually have hearty appetites, gain flesh, and enjoy themselves freely, when their attention is distracted from their subjective states; but, immediately they are spoken to concerning their delusions, the hypochondriacal self-engrossment is assumed, and they begin to lament their pitiable condition.

Cases of alcoholic delirium have been divided by Magnan into three groups, viz.:—

1. Those affected with alcoholic delirium, with easy, complete, and rapid convalescence.

2. Those affected with alcoholic delirium, of slow convalescence, with ready relapse.

3. Those specially predisposed, who have frequent relapses, and a convalescence interrupted by delirious ideas, and in which the intellectual disturbance is from the outset much more notable than the motorial.

Chronic Alcoholism.—The establishment of persistent nervous symptoms as the result of too free an indulgence in intoxicating liquors, has been for centuries recognised by the profession. Even in classic times, we find occasional allusion to such states (*Seneca*). Nor, indeed, could we conceive this to be otherwise, if we take into account the excessive vicious indulgence of the luxurious class in the later Roman Empire. Nearer our times, Lettsom has clearly demonstrated the sensory and motor troubles induced by long-continued alcoholic indulgence; but, it was not until quite recent days (1852), that a group of symptoms was formulated as constituting a distinct morbid entity under the name of *chronic alcoholism*, and to Dr. Magnus Huss, in particular, is due the credit of clearly enunciating the relationship of this important disease, which in his day was making such sad havoc among his countrymen. Northern nations have always been most susceptible to the alluring temptation of alcohol; the Russian, Scandinavian, and Scotch, being notoriously addicted to the vice. In Sweden, the consumption of large potations of raw spirit by all classes of the population (and especially of a most impure and pernicious spirit, distilled from diseased potatoes, which formed the staple commercial article), proceeded to such an extent as to demand State interference, in which the reigning family and the medical profession took a prominent part, doing much to point out the pernicious social effect of the habit, and check its further advance. The raw brandy thus consumed in Sweden was not only notoriously impure and noxious, but correspondingly cheap, and the most deleterious effects were widely apparent. It is, therefore, not surprising that the most valuable treatise upon chronic alcoholism should have emanated from our Scandinavian neighbours, and that in the classic work of Magnus Huss[*] we find detailed in no uncertain terms the ominous group of symptoms constituting a disease, whose differential diagnosis before his day had been, to say the least, most obscure and ill-defined.

Van-der-Kolk[†] dealt with alcoholism as he met with it in Holland; and later (1876), Magnan[‡] has done for France, in his elaborate treatise on alcoholism, what Huss did for Sweden; and in our own country, Drs. Carpenter,[§] Marcet,[‖] Anstie,[¶] Wilks,[**] and Parker, have, amongst many others, contributed largely to the physiological, clinical, and pathological aspects of alcoholic intoxication, and its ulterior effects upon the nervous economy. Nor must we omit to mention the highly suggestive experiments of Dr. Ogston and of Dr. Percy,[††] which gave so great a stimulus to further research into the physiological action of alcohol, and from which have directly emanated the more enlightened views now held respecting the physiological operation of this agent, its true dietetic and therapeutic value, and its operation as an incitor to morbid change.

It is unnecessary here to do more than very briefly allude to the injurious effects of alcohol on systems other than the nervous. Dr. Carpenter's "Prize Essay" did much to popularise true ideas on the subject, portraying in vivid colours, as it did, the injurious effects of drunkenness upon all the tissues of the organism. The chronic gastric catarrh; the hæmorrhagic mucous membrane; the interstitial changes in the liver and kidney;[‡‡] the atheromatous condition of the blood-vessels; the fatty changes in various organs, and notably in the heart; the functional disturbances leading to albuminuria, ascites, anasarca, gout, rheumatism, and the long list of nervous ailments; all these are familiar to any one who has paid attention to the subject. It is well, however, to recall to mind certain established physiological facts as our groundwork for further observation.

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[*] *Alcoholismus Chronicus.* Dr. M. Huss, Stockholm, 1849-51.

[†] *Influence of Strong Drinks on the Human Body*, by J. L. C. Schneider Van-der-Kolk. Utrecht, 1853.

[‡] *Alcoholism.* Dr. V. Magnan. Translated by Dr. Greenfield, 1876.

[§] *Use and Abuse of Alcoholic Liquors in Health and Disease.* W. B. Carpenter, 1850.

[‖] *Chronic Alcoholic Intoxication.* Dr. Marcet, 1862.

[¶] *Stimulants and Narcotics.* Dr. Anstie. Macmillan, 1864.

[**] *Alcoholic Paralysis.* Dr. Wilks. *Lancet*, 1872.

[††] *An Experimental Enquiry concerning the presence of Alcohol in the Ventricles of the Brain.* 1839.

[‡‡] The frequency of its action on the kidney has been denied by Dr. Dickinson and Dr. Anstie. *Medical Times and Gazette*, November, 1872.

1. Alcohol may be absorbed through the serous, mucous, or respiratory surfaces; the last fact was demonstrated by Orfila, who produced drunkenness by the inhalation of the vapour.

2. It is absorbed unchanged; and may leave the system in an unchanged form, since it has been detected by appropriate tests in all the fluids and in many of the tissues. Thus Dr. Percy, relying on its odour and inflammability, found it in the bile, urine, blood, the liver, and the brain; whilst Rudolf Masing, 1854,* and subsequently, MM. Lallemand, Perier, and Duroy detected it by the chrome test † in exhalations from the skin and in the urine.

3. As early demonstrated by Dr. Percy's experiment, it is found in proportionately largest quantity in the brain; evidencing, according to that authority and Dr. Carpenter, a peculiar "elective affinity" of nervous tissue for alcohol. ‡

4. Changes of a profound significance are induced in the blood itself at an early period, laying the foundation for the various tissue-changes which ensue, and which directly affect the well-being of the nervous centres by the immediate functional disturbances which are induced through the agency of the nutritive pabulum of the blood. Such changes are the devitalisation of the red corpuscles leading to impaired æration; to the accumulation of hydro-carbon in the blood-current, fatty specks in the red globules, whilst it causes these globules to be very slowly reddened on exposure to air.

5. Paralysis of the sympathetic system, leading also to impaired nutrition and an extravagant expenditure of animal heat.

The effect of alcohol in stunting the growth of the body is a well-known fact; animals may thus be affected when fed from an *early* age upon alcohol. At the West Riding Asylum a dog, to which alcohol had been administered for a lengthened period, not only succumbed to all the symptoms described in alcoholism in animals by Magnan (hallucination, terror, savage temper, motor tremblings, and paralysis); but the nutrition of the skeleton also became affected, so that a *notable* degree of mollities and attendant deformity ensued. Upon death, extensive fatty degeneration of the nerve cells and arteries of the cerebrum was observed. The dwarfed stature of our mining community (amongst whom excessive indulgence in drink is only too frequent) is largely due to this cause, associated with the abnormal conditions of their life and strong hereditary proclivities.

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* *Du rôle de l'Alcohol et des Anesthétiques dans l'Organisme.* Paris, 1860.

† Bichromate of potash, 1 grain; sulphuric acid, 10 grains.

‡ See "Selective Capacity of Nervous Tissues for Alcohol," by Dr. Alex. Robertson, *Glasgow Medical Journ.*, 1886.

Period of Evolution of Nervous Symptoms.—Important as it is that we should, for the sake of statistical accuracy, arrive at definite views as to the period during which alcoholic indulgence may be prolonged (ere *permanent* nervous symptoms are indicated), it is apparent, at first sight, that the question is one of extreme difficulty; and, with our existing data, cannot be answered with even an approach to accuracy. Much depends upon the *kind of drink* indulged in, the specific effects of raw spirit, wines, malt liquors, absinthe, and other drinks being too well recognised to be dealt with here; much depends also upon the *quantity taken;* the eliminating powers of the system; sex; certain diatheses (as the aguish) where the individual can take large quantities often, are all important points.

As regards *neurotic* inheritance, it is certain that, from this class of the community, drink reaps its greatest quota of the more persisting kinds of alcoholic delirium and chronic alcoholism. (See on this point, *Magnan.*)* Those *specially predisposed* to the rapid incidence of delirium upon drinking are readily recognised. We are all acquainted with friends in whom a single glass or two of wine will produce striking degrees of nervous instability; just as we recognise others in whom habitude, idiosyncrasy, or other cause permits a continuous and heavy indulgence in alcoholic drinks with but little obvious effect. It is astonishing what large quantities may thus be taken for prolonged periods with impunity; although, eventually, the nervous centres must undergo irrecoverable injury. A picture of the so-called *moderate* dram-drinker from the working-classes of Sweden, is thus given by Dr. Huss:—

> "He rises at five or six in the morning, according to the season of the year, and swallows, before going out, a cup of coffee, with a glass (2 to 3 ozs.) of brandy in it. He returns at eight to breakfast, which meal is washed down with another glass of his favourite spirits. At dinner he repeats the dose of brandy, and often adds another half glass. About five or six p.m., when his work is finished, another glass is swallowed; and supper at eight is concluded by a similar libation. During the day, therefore, he consumes from five to six glasses of brandy, or from *ten to fifteen ounces of spirit.* Such a mode of life is far from being regarded as intemperate."

Dr. Huss has known some who drank every day sixteen to twenty glasses of raw brandy. In the case of *J. C.*, the patient assured me he had frequently taken for days together twelve to fourteen glasses of raw whisky; nor did he regard this as by any means excessive.

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* "Patients specially predisposed, who, when suffering from alcoholic delirium, have frequent relapses, and a convalescence often interrupted by delirious ideas, assuming more or less the form of partial delusion." *Loc. cit.*, p. 63.

As regards *sex*, it has been affirmed that chronic alcoholism was unknown amongst women. Dr. Marcet in his interesting tables,* unfortunately, does not help us, as he excludes women from his category, because of the well-known difficulty of eliciting truthful statements in such cases. This, however, is certainly not a correct statement. Females undoubtedly enjoy a remarkable immunity from the disease, as they likewise do from general paralysis; and our experience would lead us to infer that Dr. Huss gives a fair statement of the case in his statistics, wherein he finds but sixteen women amongst a total of 139 cases of alcoholismus chronicus. The case of *M. T.* is a well-marked instance of this affection in women.

Lastly, as regards *age*. The statistics of Magnus Huss fix the *fifth decade* as comprising the larger number of cases, and the fourth decade, as presenting a smaller proportion, his figures are as follows:—

20 to 30 years of age,	14 cases.
30 ,, 40 ,,	44 ,,
40 ,, 50 ,,	57 ,,
50 ,, 60 ,,	23 ,,
60 ,, 65 ,,	1 ,,
	139 ,,

In estimating the value of this table it must be borne in mind that allowance must be made, as in all statistical tables, not only for the varying population at such period of life, but also for the prolonged period during which the agent was at work ere the malady was fully evolved; this latter consideration may have much to do with the high number of cases between the ages of thirty and fifty.

Symptoms of Chronic Alcoholism.—The functional disturbances described as present in acute alcoholism become interblended (if drinking be still persisted in) with symptoms indicative of **structural change,** the injurious action of the stimulant becomes stamped upon the organism; and the more freely the vice is indulged in, the deeper is its impress. The chief indications of such organic change are, advancing and **persistent mental enfeeblement,** with certain equally persistent **sensory** and **motor anomalies.** The general enfeeblement of the intellectual faculties supervenes slowly, but progressively; the faculties of attention, of judgment, and of comparison suffer; and memory is specially implicated. The finer sensibilities are worn off; the subject is less impressionable and sympathetic, less charitable, as well as more narrowed and selfish in his desires, his *altruistic* sentiments rapidly declining. The imaginative faculties are early affected, the higher emotional states are warped, and the whole moral nature undergoes a profound and serious change. "Apathetic, indifferent, stupefied, the chronic alcoholic bestows no attention on his person; he takes no care of his family; he is lowered in all his intellectual, moral, and social faculties, and finds himself yielded defenceless to the caprices of his instinctive appetites" (*Magnan*).* Aural hallucinations are (as in the acute form) still present, and are now no longer transitory phenomena, but often of such persistence as to constitute the chief mental symptom in the earlier stage of chronic alcoholism; the victim to such sensory disturbances suffers terribly, and not infrequently is driven to acts of desperate violence through their influence. He seeks in vain to release himself from the intolerable persecution of such voices, which blaspheme, abuse him, prompt him to commit hideous crimes, or to utter obscene and revolting language. Pursued equally by night and by day, he passes sleepless hours; often he is found sitting up in bed in a state of suspicion or of terror, declaring his enemies are beneath his bed, or outside the window plotting his destruction. At times the expressions used by his unseen foes are unintelligible to himself, are distant and scarcely audible; and he distresses his mind much in endeavouring to attach the proper meaning to such words; at other times they are loud and near, and he will attribute them to those who immediately surround him. Old associates are especially accused by the patient of thus torturing him; thus *J. M'G.* constantly heard the voice of a disreputable girl with whom he had associated; *J. J*^{1}. heard the voices of his comrades and officers of the regiment in which he had served in India; and, in fact, what Magnan states for acute alcoholic delirium is equally true for the early stage of chronic alcoholism—"The hallucinations have for their subject either the ordinary avocations, or the dominant interest of the moment."† This is but an indication of the change wrought in the last-evolved structures of the cortex, which are the first to be affected in the dissolutions induced by alcohol. Thus it was that the first patient just named (who had lived a reckless, dissipated, and immoral life of late) imagined himself to be surrounded by prostitutes who uttered the vilest language, and accused him of the most unnatural crimes; and so it was that the soldier, *J. J*^{1}., thought that he was influenced by the persons with whom he had recently associated in his military service in India.

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* "An inquiry into the influence of the abuse of alcohol as a predisposing cause of disease." By W. Marcet. *Brit. and For. Medico-Chir. Rev.*, 1862, Nos. 57–58.

The forms of chronic alcoholism which usually present themselves may be conveniently studied in three categories.

1. Amnesic cases with, or without, delusional perversion.
2. Chronic delusional insanity.
3. Alcoholic imbecility and dementia.

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* *Op. cit.*, p. 156. † *Op. cit.*, p. 34.

1. **Amnesic Forms.**—Cases comprised in this category show the earliest evidence of structural change due to the prolonged use of alcohol; they are of most serious moment, as they indicate that the border-land between disordered function and real structural change has been passed. We by no means assert that such cases cannot be relieved—indeed, great relief is frequently experienced by the subjects of alcoholic amnesia; but it is not going too far to insist that absolute recoverability is rarely (if ever) obtained from this stage of alcoholism. The more notable feature characterising this class is the peculiar failure in memory—an instantaneous forgetfulness of events which have only just occurred. Every degree is found, from slight retentiveness up to a complete, and almost immediate, abolition of the latest impression. A patient so affected forgets names, dates, and order of sequence, to an almost incredible degree. If a name not familiar be repeated over and over again, a moment's conversation will often obliterate its memory; even when told to keep the word as a test-word in mind—the recall fails, if the attention be momentarily attracted in another direction. Patients fully recognise their enfeeblement and often strive to the utmost to overcome the difficulty, such efforts eventually prove successful, and are suggestive indications as to the nature of the lesion.

J. F., aged thirty-one, a dyer's labourer; admitted in January, 1887. For four months prior to admission, he had been strange in manner, restless, and of vagrant habits. He was known to be of intemperate habits, but no satisfactory history could be obtained. Upon admission he was free from excitement and delusion; his intellectual operations were enfeebled, but this failure was evidently due to the marked impairment of memory. The following conversation, held with him in the month of February, illustrates the peculiar defect alluded to:—"What is the day of the week?" "*I don't know.*" What is the present month?" "*Don't know for certain; November, as near as I can tell.*" "How long have you been here?" *Don't remember sleeping more than one night here.*" "You gave me the same reply yesterday!" *Did I? I don't remember; if so, I suppose I have slept here longer.*" He was now told that the day was Monday, February 7, 1887, and was requested to bear it in mind. In a few seconds afterwards he was asked the date, and he replied, with some hesitation, "November, 1886; October, or November, I don't know fairly." He was quite oblivious to the names of all with whom he had been associated for some four weeks, and when attempts were made to impress them upon his memory, he rarely retained them beyond *one minute*; and if any trivial question were put meanwhile, he became immediately oblivious to the preceding impression. Old familiar airs he sings correctly, both as regards intonation and words; but any unfamiliar sequence, such as the alliterative doggerel "Peter Piper," he blunders over hopelessly and repeatedly. There is no word-blindness or deafness, no tendency to ataxic aphasia; his visual impressions are correct, as tested for colours, and general sensibility is not impaired, as registered by the æsthesiometer. The dynamometer registers for the right hand 28 kilos. pressure, and 23 kilos. for the left hand. After a residence of three months, he still affirmed that he had only slept two or three nights at the asylum. Eventually, this patient gradually improved, and a few months later he was discharged recovered.

The revivability of a former impression as a resultant depends upon several factors; thus, there are—

1. The **intensity** of the previous impression.
2. The **vigour of circulation** and **nervous energy.**
3. The **organisation** of such impressions in the establishment of associated sense-impressions.
4. The **vigour** of the faculty of **attention.**
5. The element of **time.**

Now in the case with which we are dealing, the *intensity* of the previous impression appears to be of minor importance, but the vigour of circulation and of nervous energy is decidedly at fault. Alcohol in its chronic results induces vascular paresis in various organs; and the brain and spinal cord are by no means exceptions to this rule. Then, again, the conduction along the nervous circuit is impeded in such cases, as proved by the retarded response made to sensory stimuli, *visual* or *auditory;* and this we have more reason to attribute to delay in the *sensory arc* than in the *motor arc*, or it may be due to delay in the transference from the one to the other. Such sluggish transmission can only be regarded as *resistance* in the nervous arc, and as resulting in a diminution of the effective force of the original impact at the periphery. Hence it is, that the organisation of such impressions by the establishment of associative links—*i.e.*, *the forcing of new nervous tracts* into adjacent areas (the third factor mentioned above)—becomes greatly impeded, since this greatly depends upon the vigour of the nervous current, and the vascular supply of the part.

This failure in the organisation of recent impressions was a prominent feature in the case of *J. F.*, in whom associability of ideas was most strikingly enfeebled, and an impression was received only to be the next moment obliterated; very rarely, indeed, did a recent impression act with such vigour, as to call up associated states and elicit such a response as to indicate the establishment thereby of an intellectual nexus of ideas. Such organisation is greatly aided by the *faculty of attention*, which, when directed towards the impression we tend to revive, fosters the growth of that associative process whereon a persistent and efficient memory is based. Thus it is that slight distraction of the mind, even momentarily, by directing the attention to any other line of thought will abolish the feeble tendency to organisation of the original impressions which might otherwise occur.

The faculty of attention is in these cases *itself* impaired, and the mind tends to wander aimlessly. *Time,* again, is an important element here, and in the case of *J. F.* it will be noted that any name which he was requested to bear in mind could rarely be retained for a longer time than thirty seconds, or a minute at most.

2. **Delusional Forms.**—A much larger class is comprised by those whose reasoning faculties are warped, and judgment falsified, where, in fact, delusional perversions form the most notable feature of the case. In these, as in the former class, anomalies of the sensorial and motorial apparatus may be present, and to a much more serious extent, exhibiting a far greater persistency than is observed in acute alcoholic delirium; and, in like manner, the other intellectual faculties may be involved, the power of attention enfeebled, and the faculty of recollection impaired, yet the predominance of delusions is the distinguishing feature of this class. The repeated storms which, in the acute stage, have swept over the delicate nervous arrangements of sensory or motor mechanisms have damaged these mechanisms to an irremediable extent, the emotional perturbations, the hallucinatory phenomena, ever changing and fleeting in the earlier stage, now begin to assume a more persistent, a more stereotyped form, corresponding with actual structural change in the sensorium; the intellectual aberrations evolved out of such sensory disorders, likewise lose their changeful nature, and take on a more fixed and persistent character. The exhausting character of the discharge from the highest nervous mechanism during the acute stages has left these centres in a more or less paralysed state—the energy of their cell elements is expended, or escapes in streams too feeble to produce adequate results; muscular tremor prevails; the whole life of relation is affected, and its motor activities cramped and restricted from this cause. The natural resistance to be overcome in the motor centres, although not actually increased, appears enormously disproportioned to the capacities of the volitional activities, and the subject consequently feels the encroachment of the environment which must result therefrom. The registry of this *outer resistance*, and the impotence of the will, must engender distrust, suspicion, fear, and allied depressing emotional states, whilst the illusory and hallucinatory states afford still more tangible basis for the fostering of a gloomy, suspicious, distrustful nature.

We have seen how morbid sensations may be fostered and enormously exaggerated by the persistent direction thereupon of the attention. We likewise are aware how emotional states may be aroused to unnatural intensities by the morbid tendency of the mind to dwell thereupon: so, delusional states, especially the hypochondriacal class, are fostered by the same influence, and the sense-impressions upon which they may be based are distorted to an extraordinary degree. Here, again (as indicated elsewhere), with the **decline** of **object-consciousness,** there is a **corresponding rise** of **subject-con-**

sciousness, and the conception is formed of a hostile and aggressive environment.

T. S., aged fifty-six, married, commercial clerk, admitted in January, 1873, suffering from his first attack of insanity, of fourteen months' duration. He was somewhat above the middle height, stout, muscular, and well-nourished; of somewhat swarthy complexion; hair black, turning grey; irides bluish-grey; pupils normal in size and reaction; malar and nasal capillaries dilated. Patient had been of intemperate habits, but, although often drunk, could not be called an habitual drunkard—his brother committed suicide. He had only recently been discharged from a private asylum, where he had resided for six months. He was agitated and suspicious when examined; declared he had passed a wretched night, and could not breathe naturally as the "air in the room was exhausted by some means." The instant he places his head upon his pillow he hears a whistling sound, and the voice of his late governor speaks to him; at times, the whistling and the voice are heard in the air above, and even now the voice is heard distinctly taunting him from outside the window—"Thou'st made a nice job of thyself, *T. S.*, by getting in here." He feels impelled to accuse himself of extraordinary crimes, such as murder, poisoning, and a robbery of £50,000, "although he knows it is not true." The whistling under the pillow produces "an electric current, which calls up a feeling in his hands as if he had taken morphia." When at home, voices outside his window were heard threatening his own and his son's life. Patient was a highly-intelligent man, and would talk for hours upon the subject of his delusions, which caused him much mental torture. The aural hallucinations (although present at all times, more or less) became terribly real to him at night, and deprived him of rest. He wrote a good clerical hand, and was actively employed during the first few months of his residence here; but, when left to his own resources, he invariably occupied himself in writing out lengthy epistles descriptive of the persecutions to which he was subjected. About eighteen months after admission, he made a desperate attempt to poison and hang himself.

The persistency of these delusions is evidenced by the following letter written three and a-half years after admission:—

CONSPIRACY.

Mr. F.

DEAR SIR,—I beg you will read the following without prejudice. . . . My first wife's sister's husband, who resides forty miles south of London, is the inventor of an electro-animal-magnetic machine, and other inventions, made, I suppose, somewhat similar to a camera obscura, or camera lucida, both of which or all are fixed at Leeds or Wortley (but I believe at the last-named place). Five or six persons whom I know by their voices (but there are many others I don't know) can see and hear all that transpires in the district (and to my own knowledge) within a radius of thirty miles, from Wortley; they can also tell (after having applied the electro-magnet to his head) what any person is thinking, and he is compelled and cannot avoid hearing all they say. It is impossible that mesmerism or electro-biology may be combined. The mind of the individual operated upon is affected through a material living agent, it may be through a material fluid—call it electric, call it odic, *call it what you will*, which has the power of traversing space, and passing obstacles, so that the material effect is communicated one to another. No man or woman's life is safe that they have any ill-feeling or hatred towards, so long as those infernal inventions are allowed to be practised by them. I have been operated upon for upwards of three and a-half years, by the inventor's infernal machines, by him, his, and my first wife's relatives, and others who have a deadly hatred towards me, and are intent on schemes to shorten my life, their object is to make me commit suicide, then they think they will have their own way in the disposal of all I have, say about £1,500, which I have made by railway shares, and saved out of my wages (salary) in about forty-one years. I have no doubt, whatever, in saying that these infernal inventions have been practised on me from the day I was married to my present wife, as I well remember at times, I was affected by peculiar voices, and whisperings close to my head, which were the causes of my being so very nerveless.—I am, dear sir, yours truly, T. S.

P.S.—For the last three and a-half years they have sent a continual current of electro-magnetism (or be it what it may) through my head day and night, I am prepared to prove the truth of what I have written, also can refer to parties who will verify the same. If you will come over and see me, I will give you all the information I can on the subject; also, who some of the parties are, and give you an idea where the machines, &c., are fixed. I have been twice driven from my home, from fear of being barbarously murdered. They sometimes send an electric shock through my head and say—"Take that to be going on with." . . . (here follow abusive and obscene epithets). Don't imagine I am insane because I write this from a lunatic asylum.—T. S.

Three years after admission he made another and nearly successful attempt to poison himself whilst employed in the asylum stores; having secured and swallowed a considerable quantity of *marking ink*.

No words could adequately describe the terrible mental torture to which this poor man was doomed, and although, at times (through the administration of opiates, cheerful society or employment, and conversation to distract his mind), his symptoms were somewhat alleviated—the evening invariably found him suffering, with redoubled force, from his invisible persecutors. Like most cases of this category, he exhibited the restless anxiety to discover some tangible cause for his sufferings; some plausible explanation of the diabolical means employed by his enemies. With this object he ransacked every book, periodical, and newspaper he could lay his hands upon; and eagerly questioned the medical officers as to the probabilities of mesmerism, electro-biology, witchcraft, odyle, electricity, and magnetism being the means employed. We well remember his excited expression one day when, handing us a newspaper, he indicated a passage bearing upon the telephone and phonograph, of which he had for the first time heard, and which he convincingly and triumphantly regarded as the solution to the whole mystery of his case.—*Cases of E. A. F.; G. L.*)

We have already alluded to the *resistance* offered by the environment to the activities of the organism in its life of *relation;* to the sense of proportionately increased resistance to motor energy, due to the feeble initiatory discharge; in a certain sense this applies to all the mental faculties alike. We find amongst the delusional forms of chronic alcoholism this sense of obstruction presented to every form of intellectual operation. In one, ideation is impeded—"Thought is fettered and enslaved by the unseen agency;" in another, the faculty of memory is impaired and recollection becomes painfully irksome, and this is likewise attributed to a similar power; in another, the expressive faculty of speech is restricted, and the patient declares that he is often compelled to say otherwise than he would; he will often add, "Now I am speaking *my own* thoughts; but, by-and-by, I shall be made to speak the *thoughts of others.*" One patient (*J. J*[1].) graphically describes his troubles thus :—"As I speak, the force within me will clip a word in two, and so wholly alter the meaning of what I wished to say."

In the case of *T. S.*, it will be noticed that he felt swayed by the unseen influence so far as to feel impelled to self-accusation of crimes of which he knew he was innocent. And just as *J. J*[1]. believed that he was impelled to *speak* the thoughts of others, so he was compelled to *think* as they wished him, however atrocious, however sickening, obscene, or blasphemous the line of thought. In *T. S.*, again, the revolting language to which he was doomed to listen—the horrible obscenity of speech, which he hesitated to record in writing—was a notable feature, and was equally prominent in the case of *J. M'G.* All these are instances of the enthralment of the individual faculties of the mind, leading to the sense of an invasion by an antagonistic environmental agency ; the noxious character of which is inversely proportioned to the growing sense of helplessness and incapacity of the organism. The patient, *J. S.*, died of an intercurrent affection, and, as a sad sequel to his history, his only son was admitted, at the age of forty, suffering from delusional insanity, as the result of alcoholic excess. He had had repeated seizures of delirium tremens ; had squandered a large sum of money away by his dissipated habits ; and was, on admission, the subject of phthisis, to which he succumbed in five months' time. His history was one of persistent hallucinations and gloomy delusions, in which he often thought himself accused of atrocious crimes. He was determinedly suicidal, refused food for a long time, and struggled desperately against its compulsory administration. He was surly, suspicious of all alike, could be induced to talk but little upon the subject of his delusions, and never volunteered any reference to them unless repeatedly prompted.

These cases of *delusional insanity* due to *chronic alcoholism* fall into several natural groups corresponding with the nervous centres primarily, or more prominently, implicated. Thus, there are those in whom *sensorial anomalies* preponderate, and in these the centres of special sensation, and chiefly the auditory, may be implicated ; or the centres for the organic sensations emanating from the various viscera ; or the centres for the generative organs and the sexual instincts ; all leading up to delusional perversions. There are those in whom the intellectual operations are specially, and often primarily, affected when the delusions (although often associated with aural hallucinations or hallucinatory states of other special or general sensations) are not necessarily evolved out of these. Their special character consisting in a primary change in the centres of other intellectual operations, and the resistance offered to the diminished mental energy, is registered as the immediate antagonism of a malevolent power, which has gained access to the organism. Besides these two categories there is the class already alluded to of the primary amnesic form, in which the faculty of recollection is the one more prominently or exclusively affected.

In the primarily sensorial forms we find **aural hallucinations** preponderate; and although other senses (more often those of taste, smell, and general sensation) may be likewise disturbed, in most cases the auditory disturbance is the only anomaly complained of. The phenomena observed may embrace every possible combination of articulate or inarticulate sounds. If voices be heard, they may be distant and scarcely audible, or near and loud, or in close propinquity may whisper in the sufferer's ears; they may be above, below, on the right or the left, and may be referred to casual passers-by, to animals, or to birds. Thus, one patient heard the sparrows talking to him as they flitted to and fro; another was addressed by the crows as they flew past him; but, the voice was the voice of human beings whom he recognised as his enemies. Certain French writers have alluded to **bilateral hallucinations** in which the patient hears with one ear threatening, denouncing, or revolting language; and with the other encouraging, kindly, and conciliatory words; this condition we have never met with in alcoholics. In all such cases, the malign influence makes itself felt in discouraging or alarming terms.*

A form of **visceral hallucination** is, however, often present, which is of great interest as indicative of the manner in which new but morbid groupings arise within the sensorium. It is that of the **epigastric voice,** in which a sensation felt at the epigastrium is often spoken of as a "voice" which the patient describes as not an auditory perception but still "a voice" which makes itself understood, and by which he feels himself impelled to act—this was the case with *J. W.* It would appear that a centric disturbance projected to the epigastric region is associated in some way with a disturbance impressive of the auditory centres of speech; and that the associated sensory change is referred in both cases to the same site. In fact, it may be often observed that any morbid sensation, cutaneous or visceral, will, in like manner, determine the direction from which an aural hallucination appears to emanate; both phenomena being referred centrally to the same category of maleficent agencies.

An important class is comprised of those whose characteristic delusions and illusions are those of the **sexual feelings and instincts.** A large number of alcoholics exhibit some degree of perversion of the *sexual feelings*, referred by them to an antagonistic agency; but we more especially allude to those who exhibit this perversion as the ruling spirit of their insanity. Out of such cases, the most astounding delusions are begot. A typical case who has been for years an inmate of the West Riding Asylum, refers all his morbid sensations to the generative system, which he believes to be operated upon by various agencies—electricity, poisons, caustic, red-hot iron, and elaborate mechanical contrivances worked by magnetism, which have been invented by his unseen enemies with the object of rendering him miserable and ultimately insane. Impressed with the notion that these agencies affect, not only himself, but thousands of others who are confined in asylums, he writes manuscript by the yard, revealing his feelings to the Government, and describing the various ingenious and diabolical means used for such purposes. He sketches large figures in coloured crayons, representing the human form in both sexes, delineating their anatomical structure according to his own notions, in which the generative apparatus occupy a most conspicuous position, and in which are mapped out the course of the "electric fluid," and the structures which are supposed by him to be concealed within the body for such purposes of torture.

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* "On Bilateral Hallucinations," *vide* Magnan, *Archiv. de Neurologie*, Nov., 1883.

During his relapses of excitement he recorded (on a roll of paper measuring a dozen yards in length, both sides closely written upon) his feelings and maledictory comments dedicated to his persecutors. The effusion throughout was couched in the most obscene and revolting language obtrusively exposed; in his calmer moments, no one could be more decorous and punctilious in his behaviour or conversation. The case of *J. M'G.* is allied to this; in him, also, the sexual organs were the subject of delusional perversions. Sexual hallucinations at night were frequently complained of; and his female persecutor was believed to act prejudicially upon his system from a distance through the medium of a "mirror." He, moreover, heard lewd, lascivious utterances from old associates, who imputed to him various unnatural crimes.

E. A. F., aged fifty, married, printer by occupation; a tall, powerfully-built, muscular man, somewhat prematurely aged, with a suspicious, furtive look, a dusky, sallow complexion; pupils active, the right somewhat the larger of the two: no facial or lingual tremor; no impairment of articulation. The heart's sounds were exceedingly weak, but there was no murmur, no intermission, and the pulse, which was very feeble, was regular. No insanity was traceable among his antecedents, no history of neurosis or ancestral intemperance. Patient had long been addicted to excessive drinking.

Upon examination he betrayed much nervous agitation, stared at the ceiling and walls, declaring the room was surrounded by instruments, whereby people in Leeds and Bradford could hear all that he said; certain perforations in the wainscotting he asserted were telephones; the bed he lies upon is electrified, and he even now feels the current passing through him. At his own home a telephone wire ran beneath the floor of his room, and upon placing the legs of his chair parallel with this wire, he could himself feel the electric current; people in adjacent houses constantly spoke audibly through the walls of his house; they were in intrigue with his wife, whose fidelity he distrusted. He found that she used secret signs—*e.g.*, folding her shawl in a certain manner meant that he slept, &c.; and so she communicated with his enemies.

Memory was unimpaired; his attention good; and his replies were prompt; he freely admits alcoholic excess.

Sept. 25.—Nine days after admission—still very suspicious; firmly believes his wife conceals herself in the building, and goes about looking for her. Thirty grains of chloral given nightly to relieve insomnia.

Oct. 3.—Is convinced his tobacco is poisoned; hears voices of unseen persons. The sounds issue through the ventilating outlets near the ceiling. "Cannot you hear them now?" Manner very suspicious; requests a private interview, and, when conversing, frequently expresses the fear that he is overheard.

Oct. 10.—Aural hallucinations constant during his waking hours; asserts that he hears his wife upstairs calling out, "Fool, fool, fool!" is quite convinced it is she, and if his interlocutor would but spend half-an-hour with him, he would also be convinced; heard his niece this morning, and his daughter's voice last night. He made a most violent attack upon an attendant later on.

Nov. 11.—"They galvanise all the chairs, and the current goes through me strongly."

Dec. 7.—Remarked to an attendant who opened his door this morning, "Now or never!" and followed him closely, it is not certain with what intentions.

Jan. 19.—"Hears attendants in the rooms overhead making a buzzing noise and talking;" but does not hear what they say.

Feb. 1.—No change of late; quiet, well-conducted, and sleeps well; fancied he heard a sound last night like a female voice talking to him through the ventilators of his room; and feels something like an electric current running through his right side to-day—he often feels it; fully convinced that wires convey electricity all through the wards; the voices are not sounds in reality, but "thoughts conveyed by electricity."

May 4.—Discharged "relieved," at the urgent request of his friends.

He remained fairly well-conducted and temperate for twelve months, then relapsed into his drinking habits, which produced an exacerbation of his mental symptoms, leading to a second admission eighteen months later. The hallucinations had become far more distressing; and whilst walking in the open streets, he heard voices of people miles away talking to him; suspicions against wife and children returned with redoubled force; he accuses the former of the grossest immorality; had been dangerously violent to her, and had once nearly strangled her. The night preceding his admission here, he had driven wife and children out of doors, threatening that, if they did not leave he would murder them all.

His subsequent history was but a repetition of what had preceded this attack, and he left the asylum relieved in the course of seven months.

Then we have the class of cases where disturbances of cutaneous sensibility lead to delusional concepts of a mysterious principle, known or unknown, acting upon the body. Thus, in the case of *G. L.* the room was surrounded by invisible tubes, from which issued currents of air producing electricity, and affecting his body and limbs—contorting and twisting them into various attitudes, and causing him much agony; often leaving him weak and prostrate upon waking in the morning. The unseen fluid enters his ears, and affects his brain; but his malignant enemies always keep "prudently at a distance."

A very special form of delusion, already alluded to and illustrated, becomes elaborated in such cases as that of *J. B.* Here the resistance met with by the organism appears chiefly to affect the intellectual operations, and in a very direct manner. The faculty of thought and speech become impaired, and, as the victim believes, by the direct operation of a power which has gained access to his brain; which *rules his thoughts and dictates his very utterances.* In such cases, where (as in that of *J. B*) no sensory hallucinations have been experienced, we may safely infer that the centres of the intellectual operations are primarily diseased. Thus we find attention, ideation, memory, volition in varying degrees affected in this class of cases, and delusional concepts evolved out of the resistance which is engendered by such failure.

Evolutions of Psychical Phenomena.—A case of chronic alcoholism of the purely sensorial form is obviously not one of alcoholic insanity; and it becomes an interesting and important point to trace the progress of the affection from the sensory areas pure and simple to the planes of intellectual operations—to recognise the gradual overstepping of the boundary line where intellect itself becomes involved and the case relegated to the category of the insane. With this object, let us study the nature and effects of the sensorial disturbances. The hallucinatory phenomena are, as we know, presumed to be due to certain abnormal discharges from the sensory areas of the cortex.

Nature of the Discharge.—If the nervous discharge be carefully considered we shall find it one of high tension; sudden explosive onset; rapid escape; irregular or fitful occurrence. The centres are in a state of extremely unstable equilibrium—a state of sensory hyperæsthesia prevails. When such unstable centres discharge themselves, one or more of several results may occur. In the first place, as indicated by Dr. Hughlings-Jackson, the discharging centre exhausts itself—is, for the time being (in the case of the sensory area), *less impressionable*—and has assumed a state of molecular stability. In the second place, the centres subordinate to the paralysed centres rise into uncontrolled activity. In the third place, the discharge takes a certain course and produces certain results.

(*a*) Thus, it may react along a motor tract and issue in active movement;

(*b*) It may diffuse itself in sensorial realms, producing emotional perturbation;

(*c*) It may involve nervous mechanisms subservient to the intellectual sphere of mind, and active ideation may be aroused thereby.

Whichever course it takes, whether one or the other or many, it is to be observed that the point for us to consider is the forcing of other and distant nervous tracts which are *hereby rendered more permeable* to such discharges in future.

Hallucinations a determining Factor of Morbid Ideation.—Let us revert to our original conception of the phases of object- and subject-consciousness. The sensory fibres are the channels for those pulsatile tremors which arouse the sensory cortex into activity coincident with the presentative states of consciousness. In other words, these vibratile thrills, transmitted up to the centres of general and special sensation, constitute the raw material of object-consciousness. In alcoholism we say there is a general augmentation in nerve-resistance, both in sensory and motor channels.

In the sensory nerves it is indicated by the tingling, prickling, and formication which follow the hyperæsthetic stage of the cutaneous surface—all of which phenomena are probably due to the broken-up current—the nervous impulse interrupted by augmented resistance—whereby successive shocks are no longer fused into a single impulse. Hence, excitations from the periphery do not reach these centres in the normal state; and a decline in *object-consciousness* occurs, with a corresponding rise in *subject-consciousness*. The centres themselves are in a state of hyperæsthesia, of extreme instability, and their intermittent, spasmodic discharges must take some determinate course. The discharge from the centres of special senses (which is the physical side of a hallucination) may diffuse itself along lines of least resistance in the sensory realms, discharging the numerous extremely minute nerve-granules (the reservoirs of feeling) found in these regions; and issuing in emotional states which require but slight impact for their arousal. On the other hand, if the energy of the sensory discharge be sufficiently great it will break through lines of great resistance and flood the channels of those centres which have for their psychical correlate the ideational faculties; or, overcoming the resistance of motor nerves, issue in determinate movements.

In alcoholism the *specific resistance* of the afferent and efferent fibres is augmented; the former resulting in a decline in *object-consciousness*; whilst the centric discharges *opposed* by the latter originate the depressing, emotional states associated with feelings of environmental antagonism, which is so notable a feature in this affection. Nor is the resistance ahead other than a favourable condition; for, by this means emotional states form a safety-valve for unstable discharging centres—often by motor lines, and so relieve the plane of more purely intellectual operations from the fatal results of the inrush of morbid discharges. It is only when the barrier of resistance partially gives way that the development of delusional conceptions becomes possible.

Continued discharges from these sensory areas eventually break through this barrier of resistance, but only in certain *determinate* *lines*, which become, so to speak, channelled out, and more and more pervious by the repetition of discharges along the same tract. Thus it is, that on the psychical side we find that certain hallucinations eventually beget certain determinate lines of thought corresponding thereto; that imagination becomes tinctured by the distressing hallucination and gloomy emotional background; and that, thus, the spheres of the intellectual operations become pervaded by such agencies, and strong contrasting feelings and ideas arise, overbalancing the former intellectual being; hence, the genesis of delusional states. The forcing of such tracts, or the more pervious channelling caused by energetic or oft-repeated discharge, may often be witnessed in health—note the continual recurrence, despite our inclination, of a song we have lately heard, or been impressed with; and the incontrollable tendency at times to hum or whistle over a tune which we in vain attempt to dismiss from the mind. Again, the tormenting recurrence of a line of thought, after prolonged and fatiguing mental work, which so often deprives a student of his night's rest. This persistence of sensory and ideational excitation is due to a *too-pervious channel* established, and loss of higher controlling centres; and it can only arise after discharge from the uppermost series, leaving these latter exhausted. So in these morbid states certain tracts become permeated, to the exclusion of others, by the powerful but intermittent discharge from sensorial realms, and unstable molecular arrangements are built up in the substrata of the ideational centres.

Sensory Troubles.—An early symptom in chronic alcoholism, is disordered common cutaneous sensibility, and tactile sensibility; exaltation of both usually preceding the various modifications, their diminution or abolition. Tingling, prickling sensations are often felt, and formication is especially frequent; the patient feels as though insects were crawling beneath the skin—over the thigh and gluteal regions, and gradually extending to the trunk and arms—until the feeling is at times quite intolerable: it indicates that a change is progressing in the sensory trunk and centres. Patches of hyperæsthesia are often noted, as in the wrist of the patient (*J. J*^{2}.), where tactile sensibility is greatly exalted; the site is also equally one of hyperalgæsia. The variety of hyperalgæsia, when contact causes a sensation as of burning, or of a sharp cutting edge, is also a frequent phenomenon, and in one patient the peculiar modification called by Fischer *polyæsthesia*, in which one point is recognised as two or three points, was observed. Shooting pains are prevalent in advanced cases, the pain being not infrequently associated with muscular shocks, the patient often regards them as due to electric discharges. The excessive exaltation of the sense of pain, associated with spasms and cramps, leads to ideas of the limbs being torn, wrenched off and mutilated. Later, we find numbness and blunting of general sensibility, passing into areas of complete anæsthesia (often with an extreme degree of vaso-motor paresis) which, beginning at the tips of the fingers and toes, creeps up the dorsal aspect of the limbs. An impairment of muscular sense discrimination can readily be shown to exist in alcoholic insanity; but, for the results of actual measurements of such impairment we must refer to the chapter on general paralysis (p. 298).

Motor Symptoms.—The group of symptoms described under the head of sensorial anomalies, though highly characteristic of *chronic alcoholism*, is by no means necessarily distinctive of this affection, since such symptoms may arise (individually or collectively) in other nervous diseases: the same remark applies also to the mental anomalies exhibited by alcoholics. Ordinary forms of delusional insanity often show the self-same symptomatology, the dementia of later stages of alcoholism being scarcely distinguishable from other non-alcoholic states of mental decadence. Even the *amnesic type* referred to, may be recognised as sequent occasionally to other convulsive neuroses.

It is in the motor anomalies that we find the most definite indication of an alcoholic etiology, for they especially present a distinctive group rarely, if ever, exhibited by other neuroses than the alcoholic. Perhaps too much emphasis has been laid upon the sensory and mental disturbances of chronic alcoholism to the exclusion of the motorial in the diagnostic indications usually appealed to; for, certainly, the motor group are the "tell-tale" symptoms which most clearly indicate the agencies which have been at work. It is not, however, by the grouping of such symptoms (whether sensorial, mental, or motorial) that we shall be chiefly aided in eliciting an alcoholic factor; we must chiefly rely upon the *historical* aspect of our case—the *mode of evolution* of such symptoms, and the *tendency* of the disease towards fresh nervous implications. Here, especially, do we recognise the scientific process pursued by Magnan in his classical work, when indicating the tendencies of alcoholism to pass into dementia, or general paralysis. The tendency alluded to more particularly at this phase, and upon which we do not think sufficient emphasis has been placed, is this: the morbid process due to alcoholism evolved in sensory areas *ever tends to be translated into the motor realms of the brain*, establishing necessarily a co-existent disturbance in what we may speak of as the *motor realms of the mind.* However acute, however persistent may be the sensorial disturbance, we shall always recognise a tendency towards this translation in physico-mental terms—the delusional distortions being often nothing more than mere symbols of motor enfeeblement which may not be so obtrusive a symptom to the observer.

lacksmith; admitted November, 1883. He
August, 1882, to March, 1883, and was then
go he suffered from a blow on the head by a
Upon leaving the asylum he went to work in
t of employment by a pit accident, when he
drinking very heavily, and speedily developed
olent, and talked much of the sun, moon, and
tient's father was a notorious drunkard, that
xcessive drinkers, and that two others, as well
The mother and her parentage, however, were
nt began drinking raw spirits at the age of
ttling" at a spirit-store. Since this time he has
country, often lost sight of for years by his
e end a very heavy drinker. At times he would
shocking them with his dissolute habits; secreting
nt daily, in spite of every effort to restrain him.
t the asylum he was excited, incoherent, incessantly
onal; "has the sun and moon—half the moon in his
f clothing. He had a somewhat imbecile expression,
nd talked recklessly. His face was thin, complexion
sky; the pupils were equal—consensual, reflex, and
re active. There was no defect of articulation; no
spare habit, weighing 126 lbs.; height, 5 ft. 7 ins.
ystems appeared normal.
r he still continues maniacal; grotesque, pretends to be
he patient's pockets, upsetting all the ward, and delighted
s done. He remained for twelve months an inmate, and
recovered."
for the *fourth* and last time in November, 1887. At this
of continuous maniacal excitement, usually stark naked in
on hands and knees, degraded in his habits, and requiring
was still in a jocular humour, laughed much, and gave
ies; snatched things out of the attendant's hands, grimaced
ulated much. He was very thin, his muscles flabby; facial
ted; pupils were equal and active; deep reflexes normal.
me very suddenly feeble, totters much in his gait, and is inclined
s still maniacal, but mutters more to himself; tongue somewhat

ust support himself with his feet wide apart and hands against
eet are purplish, with dark livid patches; skin looks tense,
zed; the soles of feet are blistered; both feet are of icy coldness,
mewhat rigid, but there is no genuine contracture; both knee-
mely sluggish. He takes a wide basis of support, straddles much,
ll over on his head; tongue is protruded in a slovenly fashion, first
then on the other; no labial tremor; voice loud, boisterous and
tremble much with voluntary efforts; much twitching and corruga-
-muscles, and also of facial muscles generally; frequent sudden
the body and limbs. He is still incoherent and jocular; habits
sensibility is not much impaired in the limbs.
Again improved; walks, however, insecurely; knee-jerk alm
right leg, but somewhat brisk on left side. He is garrulous, im
nd mischievous.

In this connection, therefore, the further evolution of symptoms has a topographical significance, and the morbid lesions have their site indicated in the motor-areas of the cortex, or the ganglionic masses at the base. The cortical motor-areas are especially indicated as those earliest implicated; upon them seems to fall the full weight of the toxic agency: these, the fountain-heads of volitional activities, have their energies impaired and vitiated. It is no disorder of co-ordination which we witness here; it is that of a **genuine paresis**—an absolute impairment of energy in the highest motor mechanisms. Far different is it with an allied affection of the nervous centres, also associated with psychical disturbances—general paralysis. It is characteristic of this latter disease that the implication results in disorders of **co-ordination;** in it, also, the **most special** muscular adjustments are disturbed, but in the direction of their *co-ordinate* action, and not so notably in an actual *diminution of motor energy*. In another affection also, a system-disease of the spinal cord, apt to be associated with special mental symptoms (that of locomotor ataxy), we also find the muscular power unimpaired, but the associated groupings of muscular movements vitiated.

Motor impotence, therefore, **not inco-ordination or ataxy,** is the distinctive feature of alcoholism of the motor sphere of the cerebrum. How does this motor enfeeblement betray itself? The earliest indication is usually a notable degree of fine *muscular tremor*, implicating in the first place the fingers and hand, and gradually spreading up the arm: in the next place involving the tongue, lips, and articulatory muscles generally; and lastly, extending to the foot and leg. This tremor is always more marked in the morning, and may be dissipated by a glass of spirits; if at first not obvious, it may often be brought out by prolonged extension of the arm, any slight voluntary exertion tending to establish it, when it appears as a rapid and fine oscillation of the fingers and hand. A still more important sign, however, is that of *muscular twitching*, varying from the twanging of individual fibres to a somewhat coarse fascicular contraction, implicating those muscles which co-operate in the most special forms of movement—the lips and tongue (as in speech), the *corrugatores* and *orbicularis palpebrarum* (as in emotional expression). We have on a former occasion referred to the much more frequent implication of the brow muscles as associated with disorders of attention.* Occasionally, the spasmodic action of the orbicularis palpebrarum is such as to distort the features into sudden and changeable grimaces, or the whole muscular apparatus of lips and mouth is shaken with a universal tremor; or the head and neck may be violently jerked to one side by the convulsive action of the *scaleni* and *sterno-mastoidei*, as in the patient, *J. J.*, who attributed the movement to electric currents applied by the medical officers.

* See *Brit. Med. Journ.*, Aug. 20, 1892, p. 409.

When the facial muscles are widely affected, the subject often presents a highly characteristic state. Immobile fixation of the facial muscles gives a stolid aspect to the expression, whilst silent and undisturbed; but when roused to converse, the effort at times causes such universal tremor as is seen on the eve of a flood of violent, incontrollable weeping, or upon the onset of an explosion of passion. The tremulous convulsive wave which passes over the face can be often aroused by pressing questions upon the patient, or any slight confusion. Convulsive jerking of the tongue may be complained of; thus the patient, *J. C.*, when recovering, especially alluded to the fact that he no longer found his tongue "jumping in his mouth." The patient will often recognise this muscular failure, and when speaking will put up his hand to conceal the mouth or steady its movements. *Pari passu* with this muscular twitching, paresis of the tongue proceeds, its movements become generally impeded, and speech is thick and blurred. The **stolidity** of aspect is due to defective tone; and when voluntary innervation occurs, then only is such stolidity dissipated; yet irregular **asymmetric furrowings** of the muscles of the brow are often obvious, due to irregular contractions or paralysis of the antagonistic groups. An *increased* reaction-time is a notable feature in alcoholic insanity, and although in a large percentage of cases examined the response to acoustic *as well as* optic stimuli showed this retardation, the delay was more uniformly present in the latter than the former. Taking the average range of acoustic reaction-time as $\frac{12}{100}$ to $\frac{18}{100}$ of a second, and of optic reaction-time as $\frac{15}{100}$ to $\frac{22}{100}$ of a second, in normal subjects, as given by the several authorities already quoted (p. 165), we find, on reference to the following table, that out of the twenty-six cases of chronic alcoholism, twenty exceed this maximum for acoustic stimuli, and out of twenty-four cases examined, twenty-two exceed the maximum for optic stimuli.

REACTION-TIME IN ALCOHOLIC INSANITY.

		Acoustic Stimulus. Sec.	Optic Stimulus. Sec.
G. H., . .	*Much muscular tremor,*	·14	·23
J. M., . .	*Chronic alcoholism, delusions of persecution,* .	·15	·21
H. W., .	*Chronic alcoholism, delusional insanity, violent,*	·17	·26
H. G., . .	*Dangerous, homicidal, delusions of persecution,*	·18	·21
W. W., .	*Chronic alcoholism, demented, morbus Brightii,*	·18	·25
J. C., . .	*Slight mania, hallucinations, suspicious, tremulous,*	·18	·25
E. L., . .	*Chronic alcoholism, hallucinations and delusions of suspicion,*	·19	·25
R. B., . .	*Chronic alcoholism, hypochondriasis, suspicion,*	·19	·26
J. J^1., . .	*Chronic alcoholism, hallucinations, delusions of persecution,*	·20	·26
G. A., . .	*Chronic alcoholism, delusions of persecution, violent,*	·20	·26

In this connection, therefore, the further evolution of symptoms has a topographical significance, and the morbid lesions have their site indicated in the motor-areas of the cortex, or the ganglionic masses at the base. The cortical motor-areas are especially indicated as those earliest implicated; upon them seems to fall the full weight of the toxic agency: these, the fountain-heads of volitional activities, have their energies impaired and vitiated. It is no disorder of co-ordination which we witness here; it is that of a **genuine paresis**—an absolute impairment of energy in the highest motor mechanisms. Far different is it with an allied affection of the nervous centres, also associated with psychical disturbances—general paralysis. It is characteristic of this latter disease that the implication results in disorders of **co-ordination;** in it, also, the **most special** muscular adjustments are disturbed, but in the direction of their *co-ordinate* action, and not so notably in an actual *diminution of motor energy*. In another affection also, a system-disease of the spinal cord, apt to be associated with special mental symptoms (that of locomotor ataxy), we also find the muscular power unimpaired, but the associated groupings of muscular movements vitiated.

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referred to the much more frequent implication of the brow mu
associated with disorders of attention.* Occasionally, the sp
action of the orbicularis palp brarum is such as to distort the
into sudden and changeabl rimaces, or the whole muscular n
of lips and mouth is sha al tremo
neck may be violently
the *scaleni* and *sterno*
the movement to

* Se

REACTION-TIME IN DEPRESSION AND EXALTATION—*Continued.*

		Acoustic Stimulus. Sec.	Optic Stimulus. Sec.
J. H.,	*Hypochondriacal melancholia,*	·21	·27
J. E.,	*Simple* "	·13	·27
G. H.,	" "	·19	·24
G. P.,	*Chronic* "	·22	·27
J. W.,	*Hypochondriacal* "	·23	·23
T. E.,	*Chronic* "	·26	·27
R. W.,	*Simple* "	·29	·30
S. W.,	*Climacteric* "	·29	·29
W. T.,	*Acute mania,*	·17	·24
W. W.,	" "	·17	·24
W. H.,	*Simple subacute mania,*	·18	·23
M. R.,	" "	·19	·25
W. M'C.,	*Acute mania,*	·22	·22

Increase of the specific resistance in the motor nerve-trunks may possibly explain the tremor, but that it is largely due to defective innervation of nerve-centres discharging along those tracts is highly probable, both elements taking part in the morbid state; for, even if the former (that is, the specific resistance) *be not directly augmented,* it is *relatively so increased* by a fall in the energy of the centre. A continuous contraction is thereby rendered impossible from want of a sufficiently rapid discharge from the nerve-centre; such nerve-shocks are not given off sufficiently quick; and the resulting contractions do not fuse into one tonic contraction, as in a healthy physiological state. That a pathological change occurs in the motor nerve-trunks is also indicated by the occurrence of muscular twitchings, indicative of an irritative process of the nerve-fibres affecting the motor end-plates, and terminating later on in a more or less pronounced paresis of certain muscular groups, which are then overbalanced by their antagonistic series, producing the asymmetric muscular modelling of the face already alluded to.

Muscular spasms and *cramps* are another frequent accompaniment of chronic alcoholism of the nerve-centres. They chiefly occur at night, and especially when waking from sleep. Their severity is great; the muscles of the upper and lower extremities chiefly suffer, and the resultant pain and contraction is often attributed by the subjects to the influence of their unseen foes. They speak of their wrists being wrenched round, their arms twisted and deformed, and their legs subjected to frightful torture, and they complain of aching pains and feelings of fatigue in the limbs for prolonged periods. Such cramps occur late into the history of the alcoholic, and are often at night associated with frightful dreams, when phantasms are often woven into the delusional web constituting his mental life.

The oculo-motor apparatus is by no means so frequently involved as in the allied affection—general paralysis. The pupils are often dilated and sluggish in reaction, they are seldom unequal in size, and the most advanced cases show often no impairment in the reflex adjustments apart from indications of a localised sclerosis.

Nystagmus (as the result of cerebro-spinal sclerosis) is of somewhat frequent occurrence in chronic alcoholics. Thus, in *J. W.*, continuous movement of the eyeballs occurred in a horizonal plane.

Epileptiform Attacks.—A highly characteristic group of symptoms inaugurates a later stage of alcoholism. The patient is suddenly seized with faintness, tremblings in the limbs, extreme pallor, and breathlessness; vomiting may supervene; and then slight twitching may or may not extend to a convulsive starting of a whole limb (or one side of the body), or become generally spread over all the limbs, but rarely with complete loss of consciousness. It has been conclusively shown that as regards the criminal, at least, epilepsy is a most frequent sequel in the offspring of paternal alcoholism. "In connection with this point, and also as showing the great tendency of alcoholism to produce early convulsions in the offspring, it may be mentioned that the average age for the commencement of the fits, for those epileptics who have a direct hereditary history of drink, is less by 4·5 years than for those whose parents are returned as sober." * Upon recovery a **monoplegia** or **hemiplegia** may be found to exist, and **aphasic** conditions are by no means infrequent. At times symptoms ominous of such attacks present themselves, but do not issue in convulsion or paralysis; a slight dizziness or actual vertigo, accompanied by syncopal attacks, or a mere *tendency* to faint, may be noted; or there may be pallor of face, associated with cold perspiration, while the patient sinks exhausted into a chair, but may rapidly recover, complaining only of numbness or tingling in arm or leg. The sudden onset of unilateral twitching in the face, followed by slight paralysis of that side, is of frequent occurrence in such cases; and slight "strokes" of one side of the face, or of a limb (that is, slight in *degree* of implication), and of very *transient* duration, is a prevailing symptom which recurs over and over again in this affection.

T. P., aged sixty-two, widower, a shoemaker by trade, a man of moderate height, well-nourished, but bald at vertex with scanty grey hairs around the head; the right pupil somewhat the larger of the two, both active in their reaction; the tongue protruded straight, is tremulous, superficial arteries hard, corded, tortuous, incompressible. There is no history of inherited insanity. For many years he has been a drunken, worthless character, but has not before suffered from insanity; for twelve years past his drinking habits had become most excessive, and it was apparent to all that his memory was implicated, his mental powers were becoming enfeebled, and his behaviour childish. Three months prior to his admission at the asylum, he was taken to the Union Workhouse, where he developed more active symptoms, was restless at night, developed vague fears, could not sleep, "because some one might kill him." Aural hallucinations became now apparent, and a voice distinctly ordered him to take his own life. Then it became evident that he was the subject of convulsive seizures; he denied ever having had a paralytic stroke. He was attentive in his habits, not destructive, and not violent.

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* Henry Clarke, M.D., *op. cit.*, p. 506.

The morning succeeding his admission he was restless, emotional, bursting into tears whenever interrogated; there was profound dementia. He was quite oblivious to his recent history, and did not know whether he had been days, months, or years at the asylum; in fact, all his notions respecting time were faulty. His memory failed to retain even for a few moments what he was told. His articulation was thick, blurred, and at times so indistinct as to be wholly unintelligible. In general appearance he was decidedly sottish.

Shortly after admission, he had one slight epileptoid seizure; and, with this exception, he had no further convulsive seizures for some months, when a succession of eight fits occurred one morning, leaving him exceedingly torpid and subconscious. From this state he rallied sufficiently to go about again; but was weak and tottering in his gait, and frequently staggered backwards. His dementia was more pronounced—he was very quarrelsome. One morning, shortly after this, he was seized with severe convulsions, occurring almost without intermission in the right limbs, side of face and body, and slightly in the left leg. Thirty grains of chloral, given per rectum twice in half-an-hour, caused arrest of the convulsions. Next morning he was aphasic, but no paralysis persisted in the limbs recently affected. During the succeeding three or four months, he had frequent recurrence of such convulsive seizures, with precisely the same motor distribution as in the former attack, and chloral in all cases rapidly arrested the convulsions. About three years after admission he sank in a condition of stupor, following convulsive seizures of the same nature as those already described.

(*Sectio Cadav.*) Skull-cap symmetrical, bones thicker than normal and very pale; slight adhesions of the *dura mater*. In frontal-parietal lobes of brain there is considerable wasting, most marked in the left hemisphere, and peculiarly so in the ascending parietal convolution and the boundaries of the Sylvian fissure; both hemispheres, however, have suffered much from this atrophic process. The great vessels at the base are all extremely atheromatous. The brain is pale as a whole, and its consistence somewhat reduced; the membranes are thickened, œdematous, and readily strip; the section of the brain shows much pallor of the cortex, which is notably diminished in depth in the regions already referred to as wasted; the white substance is firmer than usual, but evidently diminished in bulk, and encroached upon by greatly dilated lateral ventricles, which contain 9 ounces of fluid. A very small patch of brown induration was found, implicating the posterior portion of the right corpus striatum. The ganglia elsewhere and the cerebellum presented nothing abnormal. Whole brain weighs 1,234 grammes.

Right hemisphere, .	516 grms.	Left hemisphere, .	515 grms.
Right frontal lobe, .	235 ,,	Left frontal lobe, .	204 ,,
Cerebellum, . .	127 ,,	Pons and medulla, .	23 ,,

Heart and lungs present nothing unusual beyond hypostatic engorgement of the latter. Liver weighs 1,366 grms.; is adherent to diaphragm by tough adhesions of its capsule; its substance is dark pigmented, and very firm; in the right lobe near its upper surface is a large cyst with a distinct, white, tough capsule, and containing clear fluid. Spleen, 85 grms.; also firmly adherent to stomach; its substance congested, soft and dark. Right kidney, 163 grms.; capsule thin, strips from a smooth, pale surface, revealing numerous shallow scars; both cortical and medullary portions are much reduced—the pelvis dilated and full of fat; the organ generally is pale and unduly fibrous. Left kidney, 130 grms. Structure reduced to a narrow strip ¼ inch in diameter by a huge hydro-nephrosis—the pelvis being enormously dilated.

It must be obvious from the foregoing considerations that any division of chronic alcoholism into separate clinical groups must be a purely arbitrary measure, justified only on the grounds of convenience in clinical teaching, and in the study of the wide-spread meanderings of the diseased process; that, with greater or less psychical disturbance, either sensorial or motorial anomalies may preponderate; and that the most notable fact is the tendency to a serious degenerative process, first (but not necessarily so) implicating sensory areas, and then transferred to motor realms of the brain, implicating in its course the moral and intellectual faculties. Huss divided his cases of chronic alcoholism into six forms:—

1. The Prodromal.
2. Paralytic or Paretic.
3. Anæsthetic.
4. Hyperæsthetic.
5. The Convulsive.
6. Epileptic.

Magnan, whilst justly criticising this division as not a genuine clinical group, specially calls attention to one form—the hemianæsthetic type—which, from clinical and pathological considerations, he deems worthy of this dignity.

J. C., aged sixty, a widower, employed at his trade as currier up to a week preceding his admission, although his mental enfeeblement must have been of some duration, judging from his state at that time. It would appear that a woman, with whom he cohabited after his wife's death, had concealed his mental ailment from notice, with the object of profiting by his earnings. A maniacal outburst, however, rendered him dangerously violent, and she had to seek assistance from the Union authorities, who found him incoherent and wildly excited. He was said to have lived a temperate life; to have had no convulsion, stroke, or cranial injury; and to have exhibited failure of memory only quite recently. All these statements, however, were received with reserve, owing to the relationship existing and alluded to above. The maniacal attack may possibly have been the sequel to a convulsive seizure unperceived; be this as it may, the Relieving Officer found him noisy, incessantly talking in an incoherent strain, violent to all around, and kicking his furniture downstairs, declaring he was moving his home. He had slept but little for nearly a week.

He was a short, thick-set individual, 5 feet 3 inches high, and weighing over 10 stones; of florid complexion, with dilated malar capillaries; bald at vertex, with grey tonsure. His locomotor system appeared unaffected, and his grasping power was good; his speech betrayed no defect. His body generally was obese, but there was no distinct evidence of cardiac degeneration upon auscultation, nor were the superficial vessels notably hard or corded. His mental state was that of maniacal excitement; he was garrulous, silly in his utterances, and always irrelevant. His memory for remote events was good, but for recent events it was wholly at fault; he was utterly inappreciative of the nature of his surroundings and present condition ; attention could only be commanded with extreme difficulty, owing to his rambling off into disconnected utterances. His mood, although variable, was usually cheerful and lively; no delusion was expressed, and no hallucinations were apparent.

For a week following his admission, he was given four-drachm doses of succus conii three times daily ; the physiological effect of the drug was freely induced, but his excitement did not succumb to its influence ; and as he was weak and somewhat exhausted, the drug was discontinued. The excitement persisted for two months, during which he lost weight and looked very ill ; but, upon its abatement about this time, he again began to gain in weight, betraying, however, a very notable degree of dementia. No further maniacal outburst occurred, and he was relegated to the class of indolent, harmless, and helpless patients. A large hæmatoma auris now developed—it was believed as the result of a fall on the floor. Eighteen months after admission, he appears to have had a paralytic seizure affecting both limbs on the left side, in which common sensation was somewhat blunted and the superficial reflexes were impaired. He remained torpid and heavy for a few days, and was then allowed to get up ; he was very feeble in limb, and the left leg dragged slightly during progression. The ensuing two months were marked by rapid advance of physical and mental prostration, due to progressive atrophy of the brain ; his mental faculties were now almost wholly abolished ; nor could he stand up, although he was able to move his hands and arms freely, and with some degree of force ; if placed in the erect position, unless supported, he would double up at once. The tongue is protruded to the left, and is tremulous. At this period he was subject to threatening attacks of passive congestion of the lungs, due to failing cardiac energy.

Two years after admission, he was completely bedridden, quite helpless, and incapable of changing his position ; a bedsore formed over each great *trochanter ;* his lungs were congested at their bases, but his pulse was fair, and his appetite good, much fluid nourishment being taken ; he was utterly fatuous in aspect, and mindless Cardiac energy, however, upon which so much depends in the survival of these chronic invalids, at last became rapidly exhausted ; the lungs became greatly engorged, and he died comatose two years and four months after admission to the asylum.

It has already been shown by our remarks on the evolution of the psychical symptoms, that the invasion of the cerebrum by this agency often follows a very definite course ; and it is only in the later stages, when the wide-spread sclerosic changes in the nerve centres and the degenerative vascular lesion are most apparent, that we may get that protean aspect from a multiplicity and complexity of symptoms, which led Magnan to state that "we do not know, in fact, what symptom there is which might not be associated with chronic alcoholism under one or other of these conditions."* Such multiform symptomatology, however, does not pertain to the earlier stage of the affection, and we then cannot fail to note the tendency to a restriction of the *more pronounced* symptoms to one or the other sphere of cerebration. Thus it is often noted that the symptoms are almost exclusively sensorial, hallucinations being a most pronounced feature, whilst little or no genuine intellectual disturbance is recognised or but trivial motor ailment; other cases present themselves where the ailment, from the outset, has been a failure in the sphere of the intellect, with little or no sensorial or motorial implication; and lastly, there are those cases where the full action of alcohol appears to have been expended upon the motor sphere of the brain after a very short term of sensorial disorder. Yet, the symptoms of implication of special cerebral territories too often dovetail and overlap for any trustworthy clinical classification to be adopted; and still more frequently, if the history be one of progressive invasion of one territory after another. The more characteristic forms, however, under which cerebral alcoholism presents itself to our notice in asylums for the insane, are the following:—

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* *Op. cit.*, p. 158.

1. Purely sensorial type—(*a*) common sensibility; (*b*) visceral; (*c*) special.
2. Primary amnesic forms.
3. Premature senility, especially implicating motor areas of cortex.
4. Delusional forms with vascular lesions in basal ganglia and medullated tracts of the cerebrum.
5. Motorial types.

INSANITY AT THE PERIODS OF PUBERTY AND ADOLESCENCE.

Contents.—Evolution of Puberty and Adolescence—Pubescence as Distinguished from Adolescence—Antagonism of Growth and Development—Excessive Metabolism of Infancy—Acquisitiveness and Mimetic Characters of Childhood—Imitative Tendencies of Adolescence—Pubescent Insanity in the Female—Delusions and Hallucinations—Relapses at Menstrual Periods—Hysteric Type of Mania—Stupor Coincident with Menstrual Derangement—Case of F. W.—The Blood in Stuporose States—Case of M. A. H.—Etiology—Ancestral Influence—Periods of Susceptibility—Statistics of Hereditary Factors—Ovarian Derangements and Pubescent Insanity (A. H.)—Amenorrhœal and Anæmic States—Influence of the Environmental Factors—Percentage of Hæmoglobin in Cases of Stupor—Pubescent Insanity in the Male—Sexual Divergence—Symptoms of Pubescent Insanity—Modified Forms (J. M.)—Masturbatic and Uncomplicated Form of Pubescent Insanity—Etiology—The Moral Imbecile.

The Physiological and Psychological Evolution of Puberty and Adolescence.—Of all phases of human life, physiology deals with none more instructive than that of its critical periods. During the first and second dentition necessitated by altered conditions of life; during puberty and adolescence, when the procreative faculties are being unfolded; during the decay and obsolescence of these faculties at the menopause and grand climacteric; and lastly, during the final retrogression of senility—the physiological changes are fraught with profound interest, and in no less a degree do serious departures from normal functional activities prove suggestive to the pathological enquirer. The period of puberty, if we neglect those variations due to climatic and social influences, is usually fixed between the ages of *thirteen and fifteen for females, and of fourteen and sixteen for males.* It is emphasised by certain well-marked external signs, such as the prominence and elongation of the larynx, and lengthening of the vocal chords in the male, with a corresponding lowering of the voice an octave or more; an increased compass of voice in the female; the appearance of hair on the pubes, in the axilla, and on the face in the male; the widening of the hips in the female, and the greater vascularity of the external genitalia; an enlargement and greater activity of the sebaceous glands. These superficial evidences accompany the development of the internal genital organs, the maturation of the Graafian follicles, and the menstrual flux; whilst the galactophorous ducts of the mammæ proliferate, and true acini appear.

The genital organs are usually mature at this epoch, so far as their structure and functional activity are concerned; therefore, we may regard menstruation (which is conclusive evidence of puberty) as significant of the arrival of sexual maturity. But it is well-established that sexual maturity—that is, the *capacity for bearing children*—need not necessarily coincide with puberty, for some girls are mature before menstruation has occurred. If we have recourse to Dr. Whitehead's statistics,[*] we find that the larger proportion of cases of first menstruation occur at the age of sixteen, and that nearly 60 per cent. of the four thousand cases of puberty recorded by him, occurred between the ages of fourteen and sixteen years. I have appended to his Table the percentages for each year :—

At age of	10	years,	9	first menstruated;	or a percentage of	0·225
,,	11	,,	26	,,	,,	0·65
,,	12	,,	136	,,	,,	3·40
,,	13	,,	332	,,	,,	8·30
,,	14	,,	638	,,	,,	15·95
,,	15	,,	761	,,	,,	19·02
,,	16	,,	967	,,	,,	24·17
,,	17	,,	499	,,	,,	12·47
,,	18	,,	393	,,	,,	9·82
,,	19	,,	148	,,	,,	3·45
,,	20	,,	71	,,	,,	1·77
,,	21	,,	9	,,	,,	0·225
,,	22	,,	6	,,	,,	0·15
,,	23	,,	2	,,	,,	0·05
,,	24	,,	1	,,	,,	0·025
,,	25	,,	1	,,	,,	0·025
,,	26	,,	1	,,	,,	0·025

These Tables indicate that we may safely exclude all cases of insanity under the age of thirteen years, as not coming under the category of what we are about to consider—viz., insanity occurring in the male and female on the attainment of sexual maturity, and through the period of adolescence. The small percentage of 3·4 who show themselves sexually mature at the age of twelve, may be safely left out of consideration, more especially since cases of insanity occurring at this early age and up to fifteen are comparatively very uncommon. The term "sexual maturity" is liable to mislead; we must clearly understand by it—*procreative maturity*, and nothing more, since it by no means refers to full sexual divergence, in which the whole frame-work of the body participates, and in which the central nervous system also undergoes a profound change.

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[*] *Sterility and Abortion*, p. 46.

The period of adolescence, however, may be regarded as extending from puberty to the age of twenty-one in females, and twenty-five in males; and is characterised by most profound changes—especially by the completed development of the osseous system (*Power and Sedgwick*).

Puberty involves changes of vast moment to the subsequent stage of manhood. Anomalous conditions are but too frequently established at this epoch, which lay the foundation for future physical and mental disability. Growth is actively proceeding, and the osseous and muscular systems are adding largely to their bulk, so as to vastly increase the force and range of their activities; but with this active growth, **differentiation** and **subordination of parts** proceed until maturity is reached, and adolescence issues in full **sexual divergence.** It is a well-recognised fact that full sexual divergence is not, as a rule, ensured until the framework and its musculature are approaching maturity (*Carpenter*); and in fact, the extreme differentiation requisite for this divergence of sexual characters appears ultimately to demand a *cessation of that exaggerated nutritive activity* which prevails in the earlier periods of adolescence.* The establishment of an equilibrium in the metabolism is but one illustration of the great law of "antagonism between **growth** and **development,** which is intimately connected with the law of reproduction" (*G. H. Lewes*†). Tissue metabolism, therefore, is by no means a constant for different periods of life. The epoch we are considering is ushered in by greatly augmented activity of the nutritive functions, and affords a parallel to the conditions existing in earliest infancy and childhood.

Thus, an infant is known to treble its weight within the first year of its life (*Landois and Stirling* ‡); and from Quetelet's researches it is seen that the first three years (and especially the first year) are periods of wondrously active growth, the increase in stature being as follows :—

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* Thus Spiegelberg affirms that, "So long as the body has to provide for its own development, and consequently requires a large amount of formative material, it has no energy to spare for propagating the species. Till development has ceased, the organs which serve for that purpose remain inactive and small, and most of the important distinctions between the two sexes are absent." (*Text-book of Midwifery*, New Syd. Soc., vol. i., p. 59.)

† *Life of Goethe*, p. 355.

‡ *Op. cit.*, p. 528.

First year,	a growth of	20 centimetres.	
Second ,,	,,	10 ,,	
Third ,,	,,	7 ,,	
Fifth to sixteenth year,	,,	5½ ,,	per annum.
Twenty-fifth to thirtieth year,		Full stature attained.	

As Trousseau states—the rapidity of growth during the first three years would, if not checked, result in a gigantic stature, but, "from the beginning of the fourth year, growth proceeds more slowly up to the age of puberty, *when it takes a fresh start.*" * As at this infantile period (when the metabolism is so extremely active), every precaution is demanded to maintain the nutritive replacement of such tissue-change both in due quantity and quality, so also during puberty and adolescence, too much care cannot possibly be lavished in providing for the wants of the system—for it is at this epoch, beyond every other, that the physical and mental characteristics of the man or woman are permanently moulded or stereotyped. A parallel has been drawn by Trousseau betwixt this period of active infantile growth and the subsequent stage of adolescence, wherein he recognises analogies in morbid states at these respective epochs—the osteomalacia of adults he thus places parallel to the rickets of infancy. Excessive expenditure of nutritive forces occurs with especial frequency at the period of puberty—the lad in usual parlance is said to be "outgrowing his strength;" he may add 5 or 6 inches to his stature in a single year (*Trousseau*). As Dr. Edward Smith also states it :—"The period of puberty is associated with two classes of evils, viz., excessive development of the cerebro-spinal axis, and defective growth of the organs of organic life." † Such greatly-augmented metabolism taxes to the utmost the constitutional powers; the requirements of the very rapidly increasing mesodermal tissues are imperative and urgent; circulating albumen is rapidly extracted by the growing tissues; so that, unless a more generous diet be given, whereby such loss may be replaced, serious impoverishment must ensue. Functional disturbances as a consequence occur, whilst mal-assimilation furthers still more the vicious progress, and lays the foundation for nutritional ailments, such as tubercle, to which this epoch is so prone.

The excito-motor irritability of infancy with its jerky, spasmodic, ill-directed movements, wanting in object, wanting in power, co-ordination and skill, pass, in the growing youth, under the control of higher centres now evolving. Action is now directed to a definite purpose, and muscular activity becomes, in one form or another, the supreme pleasure of the organism; yet, such activity is still chiefly tentative, imitative, and wanting in indications of prescience and in the accomplishment of elaborate or far-reaching results. Enjoyment appears to be the purport of this vigorous and active stage of life; restless movement seems to be necessary for the expenditure of superabundant energy; and mental acquisitiveness lays up its store of facts for future use and application. The growing lad mimics the man.

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* *Clinical Medicine*, Syd. Soc., vol. v., p. 82.

† *Cyclical Changes*, p. 286.

> "A wedding or a festival,
> A mourning or a funeral,
> And this now hath his heart,
> And unto this he frames his song;
> Then will he fit his tongue
> To dialogues of business, love, or strife,
> But it will not be long
> Ere this be thrown aside,
> And with new joy and pride,
> The little actor cons another part;
>
> * * * * * *
>
> As if his whole vocation,
> Were endless imitation."—*Wordsworth.*

With the advent of puberty and adolescence all this is changed; the rapid growth of the organism is now accompanied by rapid transformations of the nervous centres, and as the parts chiefly affected are the bony framework, blood-vascular tissues, and the musculature, so should we expect a greater or less tumult in the molecular transmutations occurring at the centric expansions of the motor system of nerves; hence, the higher co-ordinating centres—the psycho-motor area—must undergo important developmental changes. Correlatively, there dawns upon the mind the consciousness of fresh motorial capabilities—the overflowing nascent energies are directed into new channels of activity, rendering new tracts of cerebral tissue permeable; and fresh motor combinations arise. An undue estimate of the subject's capacities usually exists; the imitativeness of youth declines before the *self-assurance* and originating tendencies of the adolescent. Then there crowd in upon the sensorium the impressions aroused by the slowly-developing generative organs, and the vague indefinite notions of sexual relationships gradually take form in the definite divergence of mature age; life begins to assume a reality which it formerly wanted. The mental characteristics are peculiarly of a constructive kind; and the issue may be favourable or vicious, according to the education and training then received. In some, the emotional element will be favoured, and reverie indulged in to a vicious extent may paralyse more useful and rational activities. In others, the imaginative faculties may be chiefly stimulated; the love-sick lad will pour out his plaint in verse; while girls, especially, are prone at this period to reverie and "castle-building." For the fostering of such vapid states in this class of subjects, the sensational novel of modern days appears specially designed. In the female we find the amiable virtues especially aroused, whilst in the male the dormant motor potentialities express themselves in the form of extravagant, half-developed, ill-digested plans, overweening self-esteem, and an **egoism** at once obtrusive and objectionable.

In the Female Subject.

Symptoms.—The insanity peculiar to this epoch is essentially an **acute neurosis,** not that the intensity of the symptoms is so great, as that exaltation and excitement, the symptoms of an acute cerebral process, prevail. Other forms of insanity (notably that incident to the early puerperal period) exhibit *far greater intensity* of excitement, yet acute mania prevails; and, although extremely rare, even acute delirious mania has appeared. This predominance of maniacal states over states of depression is also a feature in the insanity of the puerperal; but it is even more prevalent in the form occurring in adolescent females. Here, however, we note the influence of **sex** in modifying the type of the nervous process. Maniacal symptoms likewise predominate over depression in the male; but their frequency and intensity are notably less than in the other sex, so that the prevalence of *melancholic states* with depressing delusions becomes in this sex a far more obvious feature. This we attribute partly to vicious habits, which also to a considerable extent influence our prognosis. With this emotional perturbation we find associated much **intellectual derangement;** delusions of a definite form betray themselves at an early period of the affection; and, as we shall see later on, are, in the maniacal forms, highly characteristic. The melancholic perversions usually embrace ideas of persecution or impending trouble; notions which commonly assume the form of beliefs that the food was poisoned by relatives or friends. From a study of such intellectual and sensorial perversions, we find that delusions prevailed in one-half the cases of both sexes alike; whilst about one-fourth of either sex were subject to **hallucinations** of the special senses, the visual and aural hallucination, separately or combined, being far the more frequent.* As many as 16 per cent. of the deluded cases entertained ideas of poisoning. Religious delusions existed in a few cases, but far more frequently did their imagined troubles affect their social or domestic well-being, such as the following:—"*Robbed of all her possessions; her house in flames; mother dead and home ruined; has murdered some one and is pursued by policemen; is to be burnt alive; men concealed in her wardrobe; fellow-patients try to*

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* Or to be exact—delusions were present in 52 per cent. males, and 49·3 per cent. females; hallucinations prevailed in 25 per cent. of either sex.

murder her." These were the delusive concepts of some typical cases of this class. In most cases of this form of mental derangement, however acute be the symptoms, it will be found that excitement abates usually at an early period, even within a few days or a week of admission to asylum care. The removal from prejudicial home influences, the regular administration of good nutritious diet, and the ensurance of a due amount of sleep, cuts short the attack very rapidly. Yet this is not permanent; one or more relapses are almost certain to occur ere convalescence is finally established. All such rapid transitions from mental turmoil to calm are to be regarded with suspicion; but more especially here, where the mental derangement is itself the expression of a process closely related to the cycle of ovario-uterine excitation. At each monthly period the menstrual molimen will be associated with greater or less cerebral excitation; hence at these periods relapses are apt to occur (*M. C. W.*, p. 242).

When there is decided catamenial irregularity or suppression, when the anæmia of puberty exists, we may with confidence anticipate a relapse; nor will the more general improvement in health ensure perfect recovery in the majority of cases, until the anæmia is so far removed as to issue in the re-establishment of this function. Not that the return of the menses *cures* the insanity, but that the natural advent of this flux indicates a state of healthy function generally, and a condition of the circulating fluid which brings up the nutrition of the cerebrum to its wonted vigour. Dr. Clouston has noted this tendency to relapse in the insanity of adolescence.

> "This tendency to short, sharp attacks, with intermissions of more perfect sanity than occurs in most other kinds of mental disease, with relapses occurring one, two, three, four, and five times, and even more frequently, before recovery or dementia finally takes place, may be taken to be especially characteristic of this insanity of adolescence." *

The excitement in the less intense forms is peculiarly associated with hysteric symptoms; the subjects are, withal, often shrewd, calculating, watchful of the effect produced on the bystanders, artful, and cunning, they will sham epileptic fits or other ailments. They are often wanton, exhibit much *abandon*, are erotic in gesture, conduct, and speech, and obscenity of remark is by no means infrequent. One patient at her home, regarded as oblivious to all that her medical attendants were doing for her, enumerated afterwards every remedy tried, mentioning the dose she had heard the doctor order, and repeating his diagnosis which she had likewise overheard. Others will show much hysteric sobbing or laughter, or, assuming a childish, pettish tone, will become querulous or wildly passionate. The *extravagant* nature of the delusion often stamps this hysteric temperament. Thus such subjects will declare that they are mangled, cut into small pieces, are to be boiled alive or crucified, yet exhibit no corresponding terror. Again we often find indecent conduct and erotic tendencies associated with conditions of religious ecstasy, and boisterous, unruly demonstrativeness alternate with states of great stupor.

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* *Loc. cit.*, p. 551.

F. W., aged twenty, a married woman, had been deranged for a short period when seventeen years of age, but recovered at home She was tall, of fair complexion, muscular, but extremely pale and anæmic. Highly nervous and excitable. Regarded as of unstable mental equilibrium, a neurotic inheritance was naturally suspected, but upon close enquiry no clue was obtainable to such. Her former attack (mania) had been attributed to a lover's quarrel. About three months before admission to the asylum, she had shown a strangeness of behaviour not customary with her; had become careless of her household duties, indolent, negligent of her husband's requirements, reticent and avoiding contact with her relatives, passing her mother and others without speaking to them. She then, without any expression of definite delusion, betrayed strong jealousy of her husband, was watchful and suspicious of his movements, passed restless nights, took food scantily, her health becoming more and more impaired. Sudden outbursts of excitement supervened; she was violent and when thwarted, would try to escape by the window. During the whole of this time she suffered much from headache, and had what were described as fainting fits upon several occasions. Under medical examination her condition was that of acute mania, a strongly-marked hysteric element being associated therewith. She would roll upon the floor and sham an epileptic fit, talking incessantly much incoherent nonsense; no rational reply could be obtained from her. Later, she exhibited a tendency to intersperse religious phrases and ejaculations, with utterances of an erotic and obscene nature; her demeanour meanwhile varying from that of a fixed ecstasy to conduct betraying strongly-marked erotic features, boisterous laughter, or causeless weeping alternating with violent passion and destructive tendencies. The respiratory and circulatory systems were normal; but, as before stated, there was extreme anæmia, which accounted for the persistent amenorrhœa from which she suffered.

The tendency to **stupor** is especially marked in those cases where there is well-pronounced **menstrual derangement,** and its alternation with hysteric excitement is a frequent and interesting feature in the insanity of puberty.* In the stage of stupor complete apathy prevails, amounting at times to fatuity; the expression is stupid and demented; the pupils widely dilated; saliva dribbles from the mouth; none of the wants of the system are attended to; the hands hang helplessly, and both extremities are cold and livid. The subject is usually profoundly anæmic, a hæmic bruit may be heard over the aortic valves, or the *bruit-de-diable* over the subclavian. Such symptoms are almost invariably associated with suppressed menses, and frequently the vicious habit of masturbation prevails. Stupor, however, is not a necessary accompaniment of these menstrual derangements with pronounced vaso-motor troubles. Subjects present themselves suffering from suppression of catamenia, with excessive lividity and coldness of the hands, who are not stuporose, but excited, flippant, and erotic. The vaso-motor centres for the limbs are in juxtaposition to the cortical motor centres of the extremities, according to Eulenberg and Landois, their fibres passing apparently through the posterior limb of the internal capsule, and it is therefore to this site we must assign most probably the irritation resulting in the coldness and lividity seen in the limbs of these subjects.

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* In such instances of stupor associated with menstrual derangement, changes of undoubted moment occur in the constitution of the blood. The red corpuscles are seldom diminished in number to any notable extent, but their hæmoglobin is in all cases alike reduced in amount. In some we find the corpuscular value below half the normal, as in the case of *C. W.* (p. 386), where it is represented at ·45, or that of *R. W. J.* at ·44. The amount of hæmoglobin, as given by the several cases at page 386, fluctuates between 40 per cent. and 80 per cent. Even in the most profound stupor of *W. S.* associated with habits of masturbation (see p. 386), the percentage of hæmoglobin never fell below ·68; nor in any case of simple uncomplicated stuporose insanity have we seen the colouring-matter reduced to the extreme limits seen in cases of hæmorrhage. Thus, in the case of *M. A. M.*, the hæmoglobin registered as low as 20 per cent.

M. A. H., aged nineteen, single, a young girl of delicate physique, very thin and reduced, pale and exceedingly anæmic, suffering from her first attack of insanity of about seven days' duration. She inherited a neurotic tendency from the father's side; the great-grandmother was a paralytic; the father was insane; and the daughter was described as being of high-strung nervous temperament. She had been intelligent, and had taken an active interest in her father's business (mercantile); his late illness was regarded as the exciting cause of her attack. The patient had always suffered from catamenial irregularities, and the menses were now completely suppressed. Restlessness and insomnia were followed by hallucinations of the special senses and delusions. When brought to the asylum, her bloodless aspect was very notable, and her physical prostration great; she stared vacantly around, quite inappreciative of her position and relationships, occasionally uttered a few articulate words or disconnected meaningless sentences, and did not reply to any question asked. Her hands were decidedly cold and livid; in fact, the previous excitement had lapsed, and a condition of stupor existed; the catheter had to be regularly used before her admission. Port wine, milk and eggs, with extract of beef, were given freely. Ammonio-citrate of iron ordered twice daily and 30 grains of chloral when required at night (chloral, however, was required but seldom, as she soon obtained sleep without its aid). During the whole of the succeeding month, patient's state was one of *extreme mental torpor and apathy*; she usually sat in a half-bent posture, utterly slovenly, and negligent in her habits; saliva dribbling from her mouth; her expression vacant, fatuous; the pupils were dilated and sluggish; the extremities blue and cold; volitional effort was rare, and compulsory feeding had to be continuously resorted to. Occasionally she swayed to and fro, and gave utterance to a piteous whining or a meaningless babbling. This condition of acute dementia continued for twelve months. Her bodily health then slowly but progressively improved, and during two succeeding months she regained flesh at the rate of 10 lbs. per month; mental torpor, however, still continued, and persistent amenorrhœa was associated therewith. With this progressive physical improvement there now appeared a gradual advance to more normal states of consciousness; *but not until* *seventeen months had elapsed* did she begin to speak rationally, and about this time the catamenia appeared. She became bright and lively, but still betrayed many morbid propensities, and was mischievous, unruly, and excitable. Her progress to perfect sanity was interrupted by a short relapse; but her recovery was ensured two years after her admission.

Impulsive as these forms of insanity appear, our records show few of those desperate attempts at self-destruction, which characterise some other forms of insanity. The actual percentage of cases returned as suicidal is high (40 per cent.), whilst in male adolescents it falls to 22 per cent. This might lead one to infer that the cases were nigh as suicidal as in the form of insanity prevailing at the climacteric, when such impulses are strongly developed. This, however, is *not* the case. Hysterical forms of mania are prone to suggest or threaten such acts, but all such attempts are usually feigned and prompted by the morbid desire to create sympathy or produce effect. We must, therefore, not be misled by the fact that these cases have committed outrageous acts which seem to imply a suicidal tendency, or have frequently threatened to destroy themselves. They are not in the majority of cases suicidal in the sense that the subjects of puerperal and climacteric mania are suicidal. On the other hand, they are far more likely to turn their destructive efforts against others, and our statistics emphasise this aggressive, dangerous tendency in 48 per cent. of the female, and 55·5 per cent. of the male inmates.

Etiology.—The excito-motor exaltation of the nervous system, during the first dentition, has also its parallel in the explosive condition of the nervous centres in higher planes of cerebral activity during the evolution of the generative functions, and the sexual divergence of the epoch of early adolescence. Hence, this period is the second great trial of the constitutional powers of the subject, and is especially prone to reveal any dormant inherited vices, and call them into full activity, either as convulsive affections of the motory apparatus, such as chorea or epilepsy, or as psychical anomalies, especially of an hysteric type. We shall see further on, that the type of insanity which prevails at this period of life is essentially that of an hysterical form. We have alluded to the rhythm of nutrition, that mysterious law which dominates the evolution of all organic forms, vegetable and animal alike, as exemplified in the high-tide of infantile growth; the ebb of growing youth; the renewed flow at puberty and adolescence; and the final arrest at the maturity of manhood. Along this curve of simple vegetative growth appear the pulsations of ancestral influence. Those epochs of new developments, or the points when differentiations occur, fitting the organism for new or altered conditions of life; the ancestral energy, so to speak, adapting the organism to its altered environment—dentition, puberty, adolescence, are such epochs. They are characterised especially by the tendency to reproduce **ancestral developments**—whether normal and physiological, or only deviations from the laws of health—the new character appearing at **corresponding periods** of life in parent and offspring. Yet it must be borne in mind that ancestral vices do not necessarily reappear in the offspring *at the same period of life* as they appeared in the ancestor, and that *then*, "the transmitted characters much oftener appear before, than after, the corresponding age" (*Darwin*).* This law of inheritance has a direct bearing upon the insane heritage of adolescents, since, in them, it appears that with special frequency, we find the *ancestral vice* developed late in the life of the parent, and to be frequently an illustration of atavism. If we recall Darwin's remarks on the distinction between **transmission** and **development** of characters, we may also more readily comprehend such pathological atavism.

A remarkable persistence of any developmental vices at these periods of active life also exhibits itself, whether inherited or acquired, with which it is well to be acquainted.

In this connection, it was shown by M. Gosselin many years since, in a communication to the *Académie des Sciences*, that many special surgical affections of adolescents tend to persist, increase, or relapse throughout adolescence, but such tendency is lost at manhood—*e.g.*, ingrowing nail, valgus doloureux, suppurative epiphysal osteitis, epiphysal exostosis, subungual exostosis of great toe, and fibrous naso-pharyngeal polypi will usually defy *permanent* cure until the twenty-fifth or twenty-sixth year is reached, and temporising, therefore, is often called for until adult age is attained. †

Then again, it would appear reasonable to presume that all ancestral tendencies which are transmissible, would be peculiarly potent at those periods when the organism strives to reproduce itself; and that as ovulation occurs there would be concentred towards certain points, so to speak, the tendency to reproduce similar peculiarities, &c. The nervous system must necessarily sympathise with such conditions, and hence parental vices, and weaknesses—insanity, epilepsy, chorea—may be developed with greater frequency at this period of sexual divergence in the adolescent. That the insanity of this period *is* strongly hereditary, is indicated by the fact that 40 per cent. showed an insane heritage, and that 10·6 per cent. afforded a history of ancestral epilepsy; and 9·3 per cent. of apoplectic seizures. It is this genetic influence which so powerfully manifests itself at this period, and especially towards the end of adolescence, that forms the organised groundwork of the psychosis, and which we regard as the most important feature in the evolution of these forms of insanity; for, given an organism predisposed by inheritance to insanity, such predisposition will tell with special force at periods of reproduction and development.

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* *Descent of Man*, chap. viii. *Variation of Animals and Plants under Domestication*, vol. ii., 1868.

† See *Med. Times and Gaz.*, April, 1872.

A related law has long been recognised, viz., that variations appearing in either sex before sexual divergence is well-established, will probably be equally transmitted to either sex of the progeny; and that variations occurring late in life, when sexual divergence is complete, will be transmitted to the same sex (*Darwin*).*

Taking the 3,470 cases of insanity in our statistics, we find a clear history of family predisposition to insanity in 29·5 per cent.—for the women, 31·5 per cent., and for the men, 27·2 per cent.; hence, the inheritance by 40 per cent. in the adolescent forms is a noteworthy feature. Again, we find the neurotic inheritance generally is far above what is usual to all forms of insanity alike, as the following instructive Table reveals:—

Cases of Insanity.	Hereditary Insanity.	Parental Epilepsy.	Parental Apoplexy.	Parental Intemperance.
3,470 of both sexes, .	29·5	3·68	3·57	...
1,810 females, . .	31·5	3·37	5·35	6·6
1,660 males, . .	27·2	...	...	...
75 adolescent females,	40·0	10·6	9·3	16·0

It is, also, a noteworthy fact that the insanity incident to the male at this period of life is not (as it is in the female) characterised by a strongly-marked heredity, since only 27 per cent. male adolescents afford a history of inherited insanity, against 40 per cent. female adolescents; the inherited tendency, then, in the male sex is not above that common to all forms of insanity taken together.

On studying a series of cases of insanity occurring in the female at this period of life, we are at once struck by the paucity of cases in the earlier, as compared with the later years of puberty and adolescence. This is, of course, what we might have anticipated. The early period is one chiefly occupied with the active growth of the organism as a whole: and it is only towards the later period (when this activity of growth, subsiding, allows the generative organs to develop, and the sexual element to force itself into the mental life—when, too, tracts of cerebral tissue come to represent the reproductive system in all its relationships), that the developmental tide may issue in a stormy commotion of the nervous centres. This is forcibly illustrated by our statistics. Out of seventy-seven cases occurring from the age of twelve to twenty-one inclusive, fifty-six (or nearly three-fourths) were from eighteen to twenty-one years of age; three cases only occurred

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* *Descent of Man*, p. 232.

INSANITY AT PUBERTY AND ADOLESCENCE, WITH CO-INCIDENT MENSTRUAL DERANGEMENT OR NOTABLE ANÆMIA.

Case.	Age.	Menstrual Condition.	Remarks.
1	19	Amenorrhœa for four months.	
2	18	" of late.	
3	20	" for eight months throughout attack.	
4	16	" for eight months after admission.	Great mental stupor.
5	20	" throughout attack.	
6	20	" for six months.	Fair bodily condition.
7	14	" throughout attack.	Stout, approaching obese.
8	18	" " "	
9	19	" for five months prior to admission and persistent up to discharge.	Fair bodily condition.
10	19	" for six months prior to admission; not re-established up to four months later.	Pallid, feeble; wide, dilated pupils.
11	18	" for five months after admission.	Very pale and anæmic.
12	16	" persistent up to discharge.	
13	19	" " "	
14	20	" " "	
15	19	" for four months preceding and three months following admission.	Pupils widely dilated.
16	19	" for seven months after admission.	" " "
17	20	" persistent throughout attack.	
18	17	" of late, but precocious puberty.	Thin, pallid and reduced; pupils widely dilated.
19	20	" for six months after admission.	Very pale, anæmic; shows much torpor.
20	20	" " "	Very pale and anæmic.
21	20	" " "	
22	19	" for eighteen months upon admission.	Pale and very feeble; livid extremities; "acute dementia."
23	20	" for ten months prior to admission.	Pale and anæmic; cold livid hands.
24	16	" for four months prior to admission, persistent throughout attack.	
25	17	" history of hysteria for two years.	Phthisical.
26	19	" persists for three months after admission.	Pupils widely dilated; feeble health.
27	18	" for three months prior to and four months subsequent to admission.	Pale and anæmic.
28	20	" for three months after admission.	Wide, dilated pupils.
29	18	" for five months before admission, and persistent up to discharge.	Extremely thin and reduced.
30	20	" for six months after admission.	Pupils widely dilated.
31	20	Menstruation irregular.	
32	15	" "	Very spare and pallid; hæmic bruit; widely-dilated pupils; much stupor.
33	16	" "	Very anæmic; hæmic bruit.
34	18	" " for many months.	Pale and chlorotic.
35	20	" "	
36	19	" " and scanty.	Profound waxy pallor.
37	16	" "	Widely-dilated pupils.
38	17	" " for a short period.	
39	16	" regular.	Very pallid, weakly; great stupor.
40	18	Amenorrhœa for months prior to, and irregularity subsequent to admission.	Very pale and anæmic; developed spinal disease.
41	19	Suppression or irregularity throughout attack.	Very pale and anæmic.
42	17	Menstruation becoming for the first time established.	
43	18	Menstruation first established ten months prior to admission was suppressed or irregular throughout attack.	Extreme anæmic pallor.
44	17	Deferred puberty.	
45	19	Deferred puberty; catamenia established seven weeks after admission (recovery).	Greatly reduced.
46	17	Deferred puberty; catamenia not established.	Stout and well-nourished.

in whom menstrual derangement or notable anæmia prevailed in association with this form of insanity.

We must bear in mind the fact that every *fresh development* in the organism is attended by a correlated development in the nervous centres which represent the part; and that in no case is this more marked than when the organism attempts to *reproduce* its kind. Thus, at the menstrual molimen, when the germ is produced and shed, whatever be the mysterious influence which leads up to this effort, the ebb and flow of the developmental tide is registered faithfully in the nervous centres by a similar wave. At each menstrual molimen the sexual characteristics are more strongly emphasised by well-recognised mental states; and, *a fortiori*, derangements amenorrhœal, dysmenorrhœal, &c., are attended by deranged cerebral functions correlated thereto, and the result of discharges of grey matter. Thus most of our cases clearly show exacerbation of their mental symptoms at periods corresponding to the natural monthly term when this could be ascertained, either when the flux was present, scanty or absent. Ovarian excitation and increased functional excitation of correlated nervous centres are set up by the same influence; and this influence may expend itself sometimes on one, and sometimes on the other, system almost exclusively. It is thus we find a considerable proportion of our cases of insanity still unattended by any actual evidence of deranged ovario-uterine functions, just as amenorrhœal states may be unaccompanied by serious mental disturbance.

The development of the organism is (at such periods as we are now engaged with) strongly affected by the environment and employment; social influences, and educational systems will greatly modify the growth of mind as it does that of the body. In either case the natural lines of development may be blocked by unfavourable social surroundings, a vicious educational *rôle*, or by unhealthy occupations, which, while they undermine the physique or check its healthy expansion, often afford no food for the mental life, but dwarf its stature and cramp its unfolding energies. The periods of puberty and adolescence are peculiarly prone to suffer thus in the present day, when the struggle for existence amongst the poorer classes often demands a self-imposed bondage of body and mind, by which the conditions of life are too dearly purchased. It is amongst the poorer class, exposed to such unfavourable conditions of life, that we find the worst forms of insanity of the adolescent period prevalent.

The Blood in Stuporose States.—In this connection, it is of interest to note the constant and often profound implication of the blood in cases of adolescent and pubescent insanity, characterised by a notable degree of stupor. The following cases illustrate forcibly the impoverishment of the red blood-corpuscles in such subjects :—

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AMOUNT OF HÆMOGLOBIN IN BLOOD OF ADOLESCENT SUBJECTS (MALE AND FEMALE).

Profoundly Stuporose.

	Hæmoglobin.	Red Corpuscles.	White Corpuscles.	Value per Corpuscle.	REMARKS.
	Per cent.	Per hæmic unit.	Per hæmic unit.		
C. W., . Oct. 1, '87,	58	94·2	·20	·61	Complete relapse into stupor.
Nov. 2, '87,	60	133·8	·04	·45	Stupor of seven days' duration; catamenia scanty fourteen days ago; second time since admission; face heavy anæmic, lips blanched, pupils normal, headache.
Dec. 1, '87,	72	107·8	·10	·67	Cheerful and active at convalescent home; amenorrhœa; gums ruddy and anæmia decidedly lessened.
Jan. 11, '88,	90	115	·10	·78	During menstrual period; catamenia normal; lively, chatty, jocular; blood coagulates rather too readily.
L. E. S., . Nov. 4, '87,	70	107·2	·20	·64	Great stupor; widely-dilated pupils.
E. H., . Sep. 28, '87,	58	84·2	·40	·70	Melancholic; profound pallor.
Oct. 8, '87,	50	66	·22	·75	,, ,,
Nov. 4, '87,	58	95·8	·25	·61	Cheerful and lively.
M. A. P., Nov. 15, '87,	74	93·6	·28	·79	Intense melancholia (lactational) compulsory feeding.
H. S. L., . Nov. 2, '87,	78	102·8	·22	·76	Considerable stupor and heaviness; yet works and smiles when addressed; pupils large; no catalepsy; fair colour.
W. S., . July 25, '88,	68	100	·4	·68	Case of *profound* stupor of several years' duration ('85, '89) reported at page 188.
July 26, '88,	70	100	·25	·70	
Aug. 2, '88,	74	75	·25	·98	
Aug. 3, '88,	68	100	·40	·68	
Aug. 12, '89,	90	100	·285	·90	
R. W. J., Aug. 10, '89,	55	124	·18	·44	Quite mute, yet far less stupor than on admission; no resistance, but shrinks, expression—calm repose.
E. H., . July 29, '87,	78	97·2	·60	·80	
T. T., . Aug. 3, '87,	68	95	·40	·71	Melancholy with stupor.
Sept. 28, '87,	72	85	·20	·84	Hysteric outbursts occasional; impulsive, treacherous.
T. M., . July 25, '87,	60	115	·60	·52	Stupor; masturbates.
July 26, '87,	68	126	·08	·54	Stupor persistent.
Aug. 2, '87,	62	105	·40	·59	Less torpid.
Sept. 28, '87,	55	82	·25	·67	Cheery and much improved; far less torpid.
M. B., . Oct. 1, '87,	72	118·6	·18	·61	Relapse of hystero-epilepsy, fortnightly scanty menstruation, considerable pain.
Nov. 15, '87,	72	84·2	·22	·85	Catamenia present, much more cheery and communicative.
W. R., . Mar. 4, '88,	70	73·4	·53	·95	Melancholy; *profound* stupor.

For the condition of the muscular sense see remarks under General Paralysis (p. 298).

In the Male Sex.

Sexual Divergence.—The divergence occurring at this epoch does not proceed *pari passu* in both sexes, it appears in the male generally in advance of the female of the same age. In the boy the sexual instincts are earliest aroused. In the boy such instincts are likewise sublimated at an early period of his history, into higher emotional forms; and during adolescence the progress made in the direction of intellectual activities is more apparent, more obtrusive than in the girl. The female chiefly exhibits the **recipiency** of her nature at this epoch, the male its **projectivity** in a life of action; the former is the *receptive* organism, as the latter is the *effective* and *distributive* one. In the flooding of new areas of the cortex—in the opening up of new tracts of tissue occurring during the development of the sexual organisation in woman as in man,* the consequent differentiation is due not so much to the development proceeding entirely along a divergent tract, as in becoming more pronounced in one direction than in another, and in being more advanced in the male than in the female. The arousal of new instincts, the development of higher emotional states—vague yearnings, wide-spread sympathies, tender passions, half-understood promptings—bespeak in woman a high subjectivity, devoted, however, to the most generous ends. In man, on the other hand, the expansion of the amiable qualities is never so great; his love is from the first of a more selfish nature, and its further developments are likewise devoted to more selfish ends. More calculating, more ingenious, more inventive, his schemes and plans of action from the first must, from the very constitution of society, embrace antagonism to his fellow-man in the competition and race for life. The dependence of the one sex is notably in contrast with the self-reliance of the other. Thus Darwin, alluding to sexual distinctions, says:—

> "Woman seems to differ from man in mental disposition, chiefly in her greater tenderness and less selfishness; and this holds good even with savages. . . . Woman, owing to her maternal instincts, displays these qualities towards her infants in an eminent degree; therefore it is likely that she would often extend them towards her fellow-creatures. Man is the rival of other men, he delights in competition, and this leads to ambition, which passes too easily into selfishness. These latter qualities seem to be his natural and unfortunate birth-right." †

These considerations would in themselves suffice to indicate the divergence in type of mental ailment to which the two sexes are exposed at this period of life. Whilst the female shows a preponderating effect on the *affective* sphere of mind, the adolescent youth betrays an aberrant tendency in the *reactive* faculties of his mental being. His newly-awakened faculties, like all nascent mental products, are wondrously fresh, active, and potent; hence, naturally tending to falsify relationship from want of a due contrasting power, his powers and abilities are vastly exaggerated, and beget an unfortunate egoismus. His plots and schemes savour of the wildest vanity; whilst the self-complacent all-sufficiency with which he reveals these plans betoken the overpowering of normal contrasting experiences by the new-begot factors. Every faculty whereby he becomes a unit of power in the domestic or social circle is represented in false quantities, and a disproportionately intensified and overweening self-esteem is the natural outcome. The sexual divergence at this immature age certainly tells in favour of the gentler sex. The male adolescent has had his characteristics faithfully rendered by the amiable satire of Thackeray in the person of Pendennis, whilst his frailties have received less consideration at the hands of Carlyle.*

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* The correlation of these coincident developments is seen in the fact, that if man be emasculated, the sexual characteristics never appear (*Darwin, op. cit.*, p. 557).

† *Descent of Man*, chap. xix.

The afflative emotional states, the newly-awakened instincts, the flood of new impressions, and the sense of widely-expanding faculties, constitute a physiological stage of development which is natural to all at this period of their life. Its obtrusiveness will always be more or less noted; manly sports and exercises, with a moderate use of the intellectual faculties, will, however, do much to carry off the overflowing mental energy in a healthy channel; but of all faults, that of introspection and subjectivity at this age should be avoided. A false code of morals does much to foster this tendency, and has much to answer for in the intensification of mental anomalies in youth. Need we recall the religious asceticism of the Middle Ages as confirmatory of this fact? A transitional epoch, such as this, is surely not a suitable period for self-analysis; and this is emphasised by the well-known fact that youths, encouraged at this period by misguided parents or tutors to lead a too studious sedentary life, devoid of healthsome exercise, and to subject their mental life to a pseudo-religious training, embracing rigid introspective exercises, lapse readily into the worst forms of mental derangement, and indulge, above all others, in secret sins and sexual vices.

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* "I have heard it affirmed (surely in jest) by not unphilanthropic persons, that it were a real increase to human happiness, could all young men from the age of nineteen be covered under barrels, or rendered otherwise invisible, and there left to follow their lawful studies and callings, till they emerged sadder and wiser at the age of twenty-five. With which suggestion as a practical scheme I nowise coincide. Nevertheless, it is plausibly urged, that as young ladies are to mankind, precisely the most delightful in those years, so young gentlemen do then attain their maximum of detestability. Such gawks are they, and foolish peacocks, and yet such vulturous hunger for self-indulgence, so obstinate, obstreperous, and vain-lorious; in all senses so froward and so forward."—*Sartor Resartus*, "Getting der way."

This period witnesses the profound changes of complete sexual divergence, and the attainment of those mental characteristics which distinguish the one sex from the other. The late epoch is characterised by certain important features, which especially map it out as the earliest marriageable period free from special risks, and has, therefore, been termed the period of nubility (*Matthews Duncan*). This author shows us, that, if we compute the number of first births in newly-married women at different ages, we shall find that the greatest "initial fecundity" occurs between the ages of twenty and twenty-five. Precocious marriages expose the mother to the risks of death in child-bed, or, if she survive, predispose to the bearing of an excessive number of children.

In women, the changes occurring in the pelvic bones from puberty forward are, of course, of vital importance, and illustrate well the immaturity of the ossific skeleton for the full functions of maternity up to the age of twenty.

Symptoms.—The subject usually comes before us excited, highly elated, his attitude, demeanour and expression indicative of intense self-complacency and assurance. The excitement may be very acute, attended with continuous garrulity, incoherence and movement, yet the buoyancy of spirits is a striking feature at all times. In the more coherent states the subject, unprompted, reveals his exalted notions; talks of his acquirements as a scholar; expatiates on his skill as a workman; revels in the supposed possession of rare and much-esteemed faculties, of persuasive eloquence, of poetic talent, of wondrous vocal powers, of the gift of tongues, of artistic abilities, or histrionic powers of a high order; or his thoughts course in the direction of his muscular energies and capacities, he assumes his strength to be almost superhuman, and regards himself as a champion walker, runner, wrestler, or the like (*F. S.*, p. 329). The extravagant nature of their delusions, already alluded to when dealing with the hysteric features of the female adolescent (p. 376), is peculiarly striking also in the male subject; the *intensity* of feeling reveals itself often in short, curt aphorisms, in the tendency to antithesis; in the gestures, pantomime, pose, speech and its contents, may be recognised, more than in any other species of mental derangement, what we would elect to term the true *staccato* type of insanity.

Even if such beliefs are not definitely expressed, egoistic sentiments prevail, and are the fount from which issue extravagant schemes of action. Inventiveness, ingenuity, cunning, are all assumed by this alien being, whose mental life is awaking, though in an anomalous form, to the appreciation of the keen competition of existence. We observe a similar condition arise at a later period of life, in that fatal malady general paralysis; but here, to account for the symptoms, there is a far more profound structural alteration, which progressively becomes more and more involved, until utter fatuity and paralysis result; yet in the early stages of this disease the same egoism, the same lofty ideas of the subject's physical powers, wealth, capabilities, ingenuity and skill come to the fore.

Towards their own sex this self-assumed superiority calls forth often an aggressive conduct, an overbearing manner amounting to arrogance, which involves them in frequent disputes and quarrels. To the gentler sex their behaviour is often gallant and condescending, savouring of a precocious manliness which does not accord with their mental and physical development. As Dr. Clouston states:—"In the males heroic notions, imitation of manly airs and manners, an obtrusive pugnaciousness, and, sometimes, a morbid sentimentality were present."* And again:—"The physical appearance of the males was boyish, and of the females girlish." On the other hand, it must not be forgotten that such adolescent forms of insanity are prone to prurient thoughts and erotic promptings which make them objects of anxiety to their guardians in relation to the other sex. All the above symptoms are liable to intensification by the vicious habit of masturbation still further reducing the nutrition of the nervous centres; above all agencies does it prove most powerful in leading up to chronic delusional insanity, or into hopeless dementia.

The cases of insanity occurring from puberty to the completion of adolescence naturally arrange themselves under two categories:—

(*a*) There are those in whom **maniacal excitement** (often very intense) prevails, with the egoistic, self-laudatory state alluded to; and often alternating with conditions of mental stupor and cataleptic states.

(*b*) And there are those of a later age, in whom **delusions** are the prominent characteristic—delusions more often associated with **melancholic depression** than with maniacal excitement.

In fact, the proportion of delusional cases occurring between the ages of twenty-one and twenty-five is far greater than that which occurs between thirteen and twenty-one. It has already been remarked, that cases of melancholic depression are of far less frequent occurrence in the female than in the male adolescent, and since adolescence is completed in the female earlier than in man, age possibly has much to do with its predominance in the later adolescence of the male sex. Our statistics indicate, that of all cases of insanity apparently influenced by adolescence in men—*i.e.*, from the age of fourteen to that of twenty-five, inclusive—about one-half occur up to twenty-one years of age,* and the remainder subsequently; or, to be exact, 142 occurred before twenty-one, and 135 afterwards and up to twenty-five years of age. As in women, so in men we find that there is a rapidly increasing number of cases from the age of fourteen up to that of twenty-one.

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* *Lectures on Mental Diseases*, p. 552.

As modified by the vice of masturbation, we find the prevalence of pseudo-religious exaltation, indulgence in cant, and development of fixed religious delusions. One patient conceived himself transformed into the Almighty; another believed he was inspired by spiritual agency and could perform miraculous works; another had the gift of tongues. Then come periods of great impulsiveness (often prompted by visual and aural hallucinations), sudden ferocious violence, indecent assaults upon the other sex, and even suicidal acts of determined character, attempts at rape, strangling, drowning; these are not unusual features in the masturbatic adolescent. The type is by no means always that characteristic of the ordinary sane masturbator, for, though the physical symptoms of cerebro-spinal irritation may be equally prominent in both, yet the mental ailment borders more often on that of delusional insanity. The shy, averted look, timidity, obsequiousness, and shunning of society, may be replaced by a bold audacious bearing, a shameless confession (and even defence) of their habit, a shocking disregard of decency, and an entire absence of the sense of moral degradation. Even if the vice be concealed, the pale anæmic aspect, the dark areola around the eyes, the dilated pupil and the general atony exhibited in feeble heart and languid circulation, enfeebled motor power and disturbed co-ordination, the amnesic states, occipital headache (*Spitzka*), the vague unreasonable alarm, eccentric dislikes fostered, and unfounded suspicions, soon attract our attention.

J. M., aged nineteen, labourer; admitted June, 1882. A paternal uncle died demented; a second cousin, S. M., is at present a patient in this Institution—no intemperance. J. M. had been addicted to masturbation since the age of fourteen, and though conscious after some time of its prejudicial effects, was unable to discontinue its indulgence. During the four years preceding his admission to the asylum he grew more and more despondent, brooding ever over his Bible and Prayer-book, neglecting his work, and wandering abstractedly about the fields. This condition was interrupted by longer and shorter remissions, but finally settled into a permanency of deep depression, the outcome of which appeared, some five months before being put under care, *in a successful attempt to emasculate himself.* Immediately on its performance he had two convulsive seizures, not, however, recurring. A short period of mental improvement followed, and then he became worse than ever. Three years before this occurrence he had had a fall from a hayloft upon his head.

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* From the age of thirteen to that of seventeen there were 40 cases of insanity out of a total of 3,000.

When admitted, he was tearful and much depressed, but communicative. His object in castration was "to prevent the possibility of masturbation," but the object attained, he was overcome by fear of having "cut himself off from God, and lost his soul." This belief he maintained during his residence, but became nevertheless more slightly oppressed by it, less gloomy and despondent, and decidedly improved in some points, inasmuch as he employed himself actively, and showed sociability. Was sent to home-care at the expiration of fourteen weeks.

Without attempting any refined analysis of the multifarious groups which might be comprised under the head of insanities at adolescence, still less attempting to dignify with specific significance the varied symptoms of such groups, it will best serve the purpose of the student to direct his attention to the symptoms proper to insanity at this period of life, and to the modifying influence of vicious habits of life, *e.g.*, masturbation and drink; intercurrent affections, such as phthisis; or defective states of the blood, as in anæmia. And first, let us draw the distinction clearly between the forms of insanity arising during adolescence, and those subject to the modifying effects of masturbation.

In drawing such a distinction we must keep in mind the fact that the vice is so frequent that a pure uncomplicated form of insanity at this period of life is the exception, not the rule. As already stated, a great proportion of our adolescent cases are found to be mild cases of congenital defect, and these are, as is well known, prone to the vice.

Again, the period of life is prone to induce the habit in a case of insanity of any standing, even if the subject were not previously addicted thereto; and in the later stages of such unfavourable cases as lapse into dementia, the habit almost invariably exists.

In a pure uncomplicated form of insanity at adolescence, we find the patient in a state of sub-acute or acute excitement, with exalted self-feeling. His egoism (which is the prominent feature) is like that of the victim of general paralysis in its obtrusive aspects; there are notions of wealth, superabundant energy and power, enviable distinctions and rank, a general feeling of *bien être;* or the youth may have wondrous plans in view, exhibit restless energy, incessant scheming, yet withal he shows a frank, bold, generous bearing wholly distinct from that worn by the masturbator. He is garrulous, obtrusive, often objectionably so to his elders, yet there is nothing of a repulsive character. His egoism is ever tending outwards towards the realisation of his phantom schemes; there is no *self-engrossment and abstraction*. His egoism, again, has more self-confidence, and appears as a self-assertiveness and assurance, so well grounded in the patient's sentiments and beliefs, that opposition, dissent, restriction appear impossible, or too contemptible to be taken into account; there is not the fear, suspicion, hatred of the environment fostered by the other form. Then, again, the physical symptoms due to the vice are absent in the uncomplicated form. The recoverability, again, of insanity at adolescence is very great; the prognosis in insanity modified by onanism is very grave; in fact, the majority of unfavourable cases of the former are accounted for by the frequent lapsing into this habit. As modified by the vice, however, the mental symptoms are those of a narrow repulsive egoism, flavoured by pseudo-religious hypochondriasis, often with much shyness and reserve at first, but later on, obtrusive and unseemly (*F. S.*, p. 329). The pseudo-pietistic notions early developed, long before marked mental derangement is actually recognised by the patient's friends, are of the most cramped and selfish nature. Obstinate narrow bigotry often results in a complete intellectual famine, the patient becoming a prey to some sectarian community, which succeeds only too well in checking the due expansion of the moral nature, and in fostering the self-opinion and conceit of its victim. We find the parents often speak of such an one as of deeply-pious habits of thought and life, as eminently conscientious, as of an amiable, modest, and retiring nature, failing wholly to realise the deep-seated egoism and self-contemplative abstraction which lies beneath such natures.

With this morbid subjectivity there is often associated much timidity, unexplained dread, tremblings, frightful dreams and "nightmares," often hysteric seizures. In the intellectual sphere there is a great want of spontaneity, lack of energy and mental movement, which may border on imbecility; irritability, spasmodic temper, impulsive conduct alternate with gloom, despondency, torpor; the mood is very variable.

Then, again, hallucinations of sight and of hearing very frequently prevail; and often explain the timidity and terror of such cases. The physical symptoms are no less striking than the mental, and bespeak the wide-spread exhaustion of the cerebro-spinal centres. Such symptoms are—the anæmic aspect, associated with general atony; the dilated pupil; the languid circulation and vasomotor paresis; cold blue extremities, a sense of weakness in the lower extremities, and a slightly ataxic gait, often swaying and inco-ordinate movements.

We thus see that the symptoms indicative of the psychosis, incidental to this period of life, are far different to those aroused by the vice of masturbation. The egoism and afflative state of the maniacal adolescent are readily recognisable, but their symptoms undergo varied modification and intermixture upon addiction to this vice. Thus, when we find the adolescent, instead of improving rapidly, makes several partial recoveries only to relapse, and especially when such relapses are towards mental torpor with general lack of muscular energy and vascular tone, we at once suspect addiction to this vice. So when we find the averted glance, the widely-dilated pupil, the expression and demeanour indicative of effeminate self-engrossment, and delusions based on the sexual instincts, indications of sexual perversions or intensified egoism, we naturally look for a similar origin. Aural hallucinations, timidity, distrust, loss of self-confidence with this concentration of the self-feeling are all harbingers of the same vicious habit.

Etiology.—A very large proportion of the cases of acute excitement are constituted by congenitally weak minds, and the number of such cases which precede the age of twenty-one is nearly double that of those which follow; in fact, the age of puberty and adolescence is peculiarly the trial-period for subjects of congenital defect—then, if at all, will their deficiencies become notably prominent. This remark does not necessarily apply to cases wanting in intellectual aptitude, but rather to those with the defective moral control which characterises so many of our cases of congenital weakness. The whole moral being, as we have seen, is subjected at this period to revolutionary changes through the incoming of new sensations and the turmoil produced by this interpenetration of the old self; new sentiments spring into life, fresh-begotten emotions (redundant in energy) tend to further overthrow canons of belief; and the judgment is strongly swayed by such overbalancing factors. At this period, if at any time, is a duly-balanced moral control necessary to the well-being of the subject. How often it fails is too flagrant a fact to be dwelt upon. How much of such failure is due to immature and narrow systems of education, to vicious and cramping customs of life, and to injudicious parental training is also only too apparent. Of the most vital importance is it that the lines of development of the moral nature at this critical epoch should be watched with the greatest interest, and that the parental and tutorial guidance should be of the most enlightened and prospective kind, to insure the due integration and elaboration of the chaotic mass of impressions incrowding at this stage of mental evolution. It is out of such misguided states arise the religious fanatic, the sordid sensualist, the repulsive masturbator, the nerveless sentimentalist, and the vicious and impulsive characters who are to the end moral wrecks, bearing witness through their lives to the violence and tyranny of the adolescent storm. It is the epoch of great moral convulsions, which in the insane is accountable for those extraordinary delusional concepts of a religious character, which (even in the insane) have so bizarre an effect. The ideas of being crucified or of being subjected to martyrdom of a revolting kind, of being transformed into the Almighty, exemplify the kind of notions which readily spring into life at this epoch in those who become alienated. Such conceptions, it may be noted, would be scarcely possible at a much earlier age; they deal with the subject-matter of late periods of development, the material of religious doctrines and sentiments, and therefore indicate an early denudation of evolving mind. Prior to the mental commotion of puberty these *moral imbeciles* have, maybe, shown an aptitude for learning, a brightness of intelligence proportionate to their age, and little (beyond an ill-governed passion, or vicious or cruel tendencies) to indicate the approaching danger.*

When the sexual instincts are aroused at puberty, their dwarfed *morale* renders them easy victims to the vice of masturbation, which, perhaps, is the best criterion of defective moral control. If persisted in, it no longer remains the symptom of a mental defect, but the prolific source of a deepening malady of the nervous centres, whereby the mental affection is itself coloured. Masturbation as a *symptom of disease*, is, of course, prevalent in insanity at all periods of life; but adolescence is the epoch, especially, when its indulgence is apt to be the exciting cause of a grave developmental malady, which otherwise might have been tided over. We have on more than one occasion watched the advent of puberty in the successive members of a highly-neurotic family where insanity, drink and apoplexy had been the ancestral curse, and have seen one after the other succumb to this epoch of their life, the vice being successively engendered as the sexual instincts came to the fore. There are few physicians who do not meet with numerous instances of this class.

The explosiveness of nerve-tissue in the imbecile is a characteristic feature of their case, and we can conceive no condition more likely to issue in the impulsive forms of insanity than that of a vicious imbecile arriving at the period of puberty and a victim to a perverted sexuality.

Prognosis.—Of all types of insanity that occurring at the puerperal period is one of the most recoverable (80 per cent.), yet the recovery-rate is nearly as high for the insanity incidental to adolescence; in the female sex the mania runs a course of some months, usually marked by one or more relapses, but one half of the cases recover by the seventh, and nearly three-fourths are well by the tenth month. (*See Chart B.*) It is far otherwise with the same affection amongst men; in them, where (as already stated) depression is often largely present, the prognosis is far less favourable, and a wide margin must be allowed for partial recoveries, chronic incurables, and fatal cases. On comparing the percentage of recoveries in puerperal, adolescent male and female cases, the above statement is fully verified, thus:—

	Puerperal Cases.	Adolescent Females.	Adolescent Males.
Recovery-rate per cent., . .	80·0	73·3	58·4

A glance at the following table of results of treatment in either sex, will indicate in no uncertain terms the more unfavourable nature of this form of insanity in the male sex:—

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* See also on this subject Dr. Hack Tuke. *Psychological Medicine:* Art., "Pubescent Insanity."

	Recovered.	Relieved.	Remained as Chronic.	Died.
Female adolescents per cent., .	73·3	7·6	13·5	5·4
Male adolescents ,, .	58·4	14·4	17·3	9·7

The unfavourable cases form 26·5 per cent. of the total number of cases in females, and 41·4 in males.

We have already alluded to the actual recoverability of this form of insanity in its relationship to sex; it will also repay us to observe more particularly the duration of the attack up to cure in both sexes; in other words, the recovery-rate as affected by *time.* (*See Chart B.*) If for this purpose we glance at the chart of recoveries first as regards the male adolescent, it will be evident that during the first six weeks but ten cures will result out of a total of one hundred and sixty-two; during the next fortnight an addition of ten recoveries just doubles this number, and then for the third, fourth, and fifth months a rapid increment of cures—viz., seventeen, fourteen, and twenty-five respectively; so that the summit of the curve is reached at the fifth month,* *by which period nearly one-half of the total number of cases have recovered.* Then a sudden drop midway occurs for each month from the sixth to the ninth inclusive—*i.e.*, from ten to twelve cases for each month respectively. From this time up to twenty months the monthly curve once only rises above four, being usually much lower, and a few rare and unexpected cures occur (as in other forms of insanity) at later periods still.

Now the curve of recovery for females differs considerably from the foregoing, being less abruptly broken, being more uniform and sustained in the early half of the period of recoverability,† and exhibiting beyond this but one abrupt elevation; also by the critical period, if by this term we may so name the period of greatest recoverability, occurring from the fourth to the seventh month, and not as in the male subject from the third to the fifth. From this it results, that whereas one-half the male recoveries are established by the fifth month, *nearly seven months elapse ere a proportionate number of females recover.* The second abrupt rise of eleven cases, and as sudden a fall shown at the tenth month in the female chart, reproduces in a modified form the sustained recovery-rate shown between the sixth and ninth months in male adolescents. From such a chart we might augur that the chances of recovery, apart from any specially-favourable points inherent in the case, are equally good between the fourth and seventh months from onset; that if from some unfavourable element in the nature of the case recovery does not then take place, a further hope may be enter-

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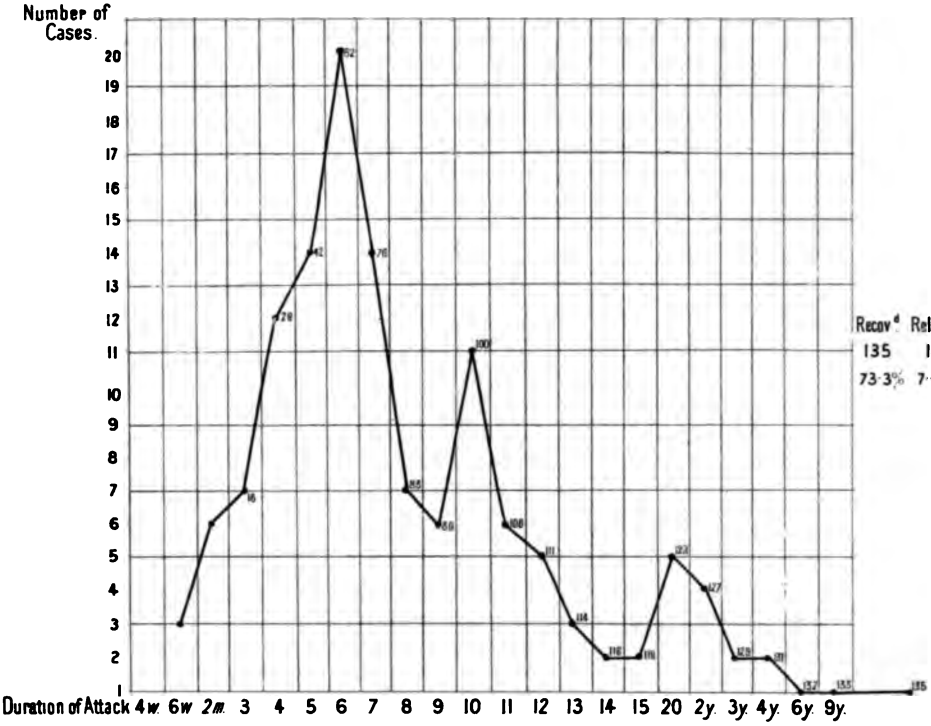
* It must be remembered that the period dealt with in these charts is that between *actual onset of insanity* and recovery.

† The period of recoverability may be arbitrarily fixed at twelve months from the onset of the insanity; the few recoveries subsequent to this date not militating strongly against the utility of this doctrine.

CHART I

CHART of RECOVERIES in FEMALES.

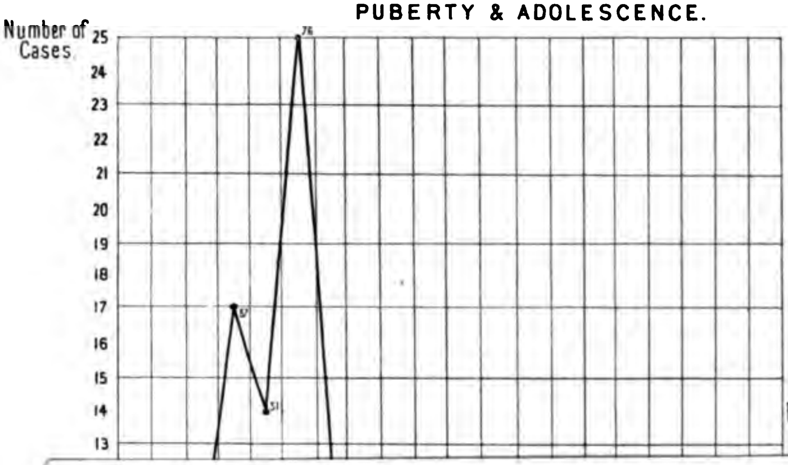
PUBERTY & ADOLESCENCE.



Recov^d	Reliev^d	Died. R
135	14	10
73·3%	7·6%	5·4%

CHART of RECOVERIES in MALES.

PUBERTY & ADOLESCENCE.



Recov^d	Relieved	Died.	Rem
162	40	27	

tained of recovery at the tenth month, beyond which the chances are greatly reduced; and, also, that if a male adolescent is not included in the favourable list of cures up to the fifth month, we may still hope on with reasonable expectation of recovery to the ninth month, beyond which the case must be regarded as assuming a serious character, and the outlook is certainly ominous.

Not only are the cases prior to the age of twenty-one more often characterised by excitement, but the type of insanity then prevalent is distinctly a more recoverable form; the recoveries in the earlier contrasting with those in the later period, as ninety-one to seventy-one. In other words, if we group together the partial recoveries, the fatal cases, and the chronic remnant as the *unfavourable class*, we shall find that such a class constitutes 31 per cent. of the acute forms of insanity, and 49 per cent. of the melancholic forms; in fact, the chronic cases are double, and the partial recoveries ("relieved") more than double in the melancholic, that which obtains in the maniacal forms.

Treatment.—The simpler forms of hysteric excitement occurring at this period often do not call for other than moral and dietetic measures; removal from the home circle and possible sources of irritation to entirely novel relationships, the administration of a due amount of aliment, regular habits of life, and means to ensure sleep will often suffice to effect a cure. Nourishment should be given in an easily-assimilable form, its nature dictated by our knowledge of the systemic wants at this developmental phase of the patient's life. The secretory and excretory organs should be especially attended to, liable as they are at these periods to derangement and sluggish action. Sleep should be secured by out-door exercise, active employment, commensurate with the patient's powers of endurance within the limits of absolute fatigue; sedatives should be studiously avoided. It is only when prolonged insomnia persists for several nights together, in spite of the above measures, that sedatives are admissible, and then a single dose of chloral, sufficiently large to ensure absolutely the desired amount of rest, may be given; its frequent administration for this purpose is to be strongly deprecated. In such cases as inherit a strongly-neurotic temperament, and in which the cycle of developmental change has not resulted in much physical over-strain and wear and tear, large doses of potassium bromide (30 to 60 grains three times daily) may be administered with decided benefit so long as a due amount of wholesome food can be taken. Most often we have to deal with the menstrual irregularities of this epoch and its attendant anæmia, our subject having succumbed to the developmental wave; the nutritive and assimilative capacities having been overtaxed by the exaggerated demands of the growing organism. Here a strictly-hygienic regimen should be carefully and persistently enforced, such as out-door exercise and the spinal *douche* or sponge-bath. Iron, especially the carbonate, should be given in pill or mixture. It is well to vary the form of iron, occasionally administering it in the form of iron and aloes pill; or as the ammonia-citrate; or as the sulphate of iron in combination with extract of nux vomica and rhubarb in pill; or again as the compound syrup of the phosphates with malt-extract and cod-liver oil.

INSANITY AT THE PUERPERAL PERIOD.

Contents.—Symptoms—Predominance of Mania—Intensity of the Morbid Process—Obtrusive Sexual Element—Hallucinations—Delusions of Suspicion—Prevalence of Suicidal Feelings—Etiology—Susceptibility of the Puerperal Period—Illegitimacy and Puerperal Insanity—Frequency in Primiparæ—Condition of the Blood—Diminution of Hæmoglobin—Prognosis—Treatment—Insanity of Pregnancy—Relatively Infrequent—Primiparæ show no Special Liability—Symptoms—Recoveries.

Symptoms.—The onset may be absolutely sudden, following upon delivery; but more frequently the development is gradual, being preceded by evidence of nervous exhaustion, until the fully-matured disease bursts out in all its fury at the end of the first week succeeding labour. The patient suffers from early insomnia, becomes restless, fidgetty, unnaturally garrulous; she exhibits a waywardness not customary, takes strange and unreasonable dislikes, especially towards her husband, or refuses to have her infant brought to her. There is a furtiveness of glance auguring a suspicious state of mind, a startled look on the slightest sound, or even intolerance of light. All her relatives observe a change of disposition, and perhaps attribute it to a wilful temper merely, but the pulse becomes hurried and small, the face pale and haggard, the eye startlingly bright. She cannot be induced to sleep, or sleep is broken by disturbing dreams, from which she starts up in bed rambling in disconnected utterances. Then come hysteric outbursts, extravagant conduct, and all the features of an acute maniacal attack. The presence of delusions and hallucinations declare themselves, she shouts aloud to imaginary persons, listens to their voices, rejects her food with repugnance, declaring it to be poisoned.

Maniacal excitement usually characterises these outbursts; out of 68 puerperal cases as many as 45 or 66 per cent. suffered from **mania,** whilst states of **depression** prevailed in 23 patients or 33·8 per cent. If we associate with these the cases which, originally puerperal, had been allowed to suckle their infants for some time after symptoms of mental alienation had been observed, we get from a total of 111 as many as 74 or 66 per cent. as subjects of mania, and nearly the same proportion, 30 per cent., as subjects of melancholia.

The mental affection, then, at this stage is essentially an **acute maniacal state,** in which there is *intense excitement, great incoherence, continuous garrulity, and a dangerous explosiveness,* which may issue in most desperate impulsive conduct; there is, moreover, a special proclivity to indulgence in *obscene* language, indecent exposures of the person, and genuine nymphomaniacal states; or the deep-rooted erotic feelings may, partially controlled, reveal themselves in the sudden gestures, or sensual glances, or prurient demeanour in a less obtrusive manner.

There is, despite the adverse view of high authorities upon this point (*Gooch, Marce, Foville*), abundant evidence in support of the view that this sexual element stamps the insanity of parturition and the *early* puerperal period with features which demand special attention; but whether such features should exalt the mental affection into a distinct nosological entity is very doubtful, and, in our opinion, unjustifiable from considerations already appealed to.*

Hallucinations.—Visual and aural hallucinations, or both combined, occur to the almost complete exclusion of other forms of sensory disturbance; in fact, 17 per cent. of our puerperal cases exhibit such anomalies—the average for the total admissions in all forms of insanity being 29 per cent. This is not so high an estimate as that of Dr. Clouston, who also assumes the aural to be the more frequent; in our experience they occur in about equal frequency.†

Delusions.—Quite 63·2 per cent. showed obvious delusions of very varied character, but chiefly tending towards ideas of persecution, the patient believing herself the victim of intrigues at the hands of her nearest relatives—her husband, children, her former friends and neighbours; her life, or that of her children, is threatened, or some terrible tragedy is being enacted; in two cases, the house was believed to be haunted. Ideas of poisoning are prominent features, the food being frequently rejected upon this plea. Another not infrequent delusion was that of a sexual nature; the husband's fidelity was called in question, or there were ideas that men entered her bedroom at night for illicit purposes; one patient believed herself to have been confined of twins who were falling into a canal. In five cases the subject believed herself eternally lost, forsaken by God, and given up to the machinations of the evil one. This delusion that the soul is lost was as frequent as in forms of insanity of a more melancholic type, occurring later on during lactation; in fact, religious delusions were frequent. Hypochondriacal delusions were not observed in a single case.

To take a brief summary of some of the more important hallucinations betrayed by puerperal cases :—

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* See also on this point Dr. Sankey's *Lectures on Insanity*, pp. 128, 195.

† Vide *Lectures on Mental Disease*, p. 507.

Faces besmeared with blood peep through the windows; spirits hover around; angels and devils surround the bed; the patient's deceased mother confronts her, and mysterious lights flit about the room; voices are heard; the form of the evil one appears; or they shout aloud to imaginary voices; sounds are heard, interpreted as conspirators beneath the building; or a voice within prompts her to suicide.

From these considerations it will be obvious that the general tone of feeling is that of **distrust** and **suspicion,** implicating her conjugal relationships, her friends, and former associates, or as affecting her moral well-being; or utter failure of self-confidence and delusions, sometimes of a most harrowing description, basėd thereupon.

Suicide.—We have alluded to the **explosiveness** of the disease, and the tendency to impulsive acts is a most notable feature in insanity at this period. Attempts to murder the offspring have been frequently recorded, and no woman suffering from this form of insanity should be brought into close relationship with her children. About 25 per cent. presented *active suicidal propensities;* but nearly double this proportion, or 47 per cent., were impulsively dangerous to those around them. The suicidal impulse was often prompted by delusion; thus, one patient believed her husband wished to cut her throat, and, consequently, sprung from her bedroom window. Another leapt from her window under the impression that her husband had just murdered his two children, and under similar impressions. One case tries to end her days by a desperate attempt at strangulation, and another by cutting her throat.

The various forms of mental disturbance found at this period may be thus classified in the order of their frequency of occurrence :—

Acute mania, ...	31	cases.
Melancholia, with delusions, ...	13	,,
Mania, with delusions, ...	7	,,
Simple mania, ...	7	,,
Simple melancholia, ...	8	,,
Acute melancholia, ...	1	,,
Melancholy, with stupor, ...	1	,,
	68	,,

Etiology.—It would be indeed strange if, at a period embracing such revolutionary changes as are comprised in the onset of labour and the first half of uterine involution,* the mental stability was not endangered beyond the average usual at the same period of life in the non-parturient. So numerous are the novel relationships into which the nervous system then enters, and so powerful are the new agencies brought to bear as excitants to morbid reaction, that subjects hereditarily predisposed to insanity, must necessarily incur imminent risks at this crisis of their history. As Burrows says:—"Gestation itself is a source of excitation in most women, and sometimes provokes mental derangement, and more especially in those with a hereditary predisposition."*

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* The term "puerperal mania" is arbitrarily assigned to the mental derangement occurring during the *first six weeks of the puerperal state;* involution of the uterus being usually not complete for three months.

Let us consider what are the peculiar circumstances which favour such issues. First, there is the mental transformation incident to this latter period of gestation; the arousal of maternal instincts, especially for the first time, is frequently associated with unstable states of nerve-centres, issuing in introspective states, exalted self-feeling, voluminous emotional waves, vague fears of impending troubles, and often hysteric outbursts. Such conditions must be attributed to the eccentric irritation of the gravid uterus, as well as to the deteriorated state to which the maternal blood has succumbed; and, unless the depurative processes become more active, nutritional anomalies are liable to arise, producing undue nervous instability during gestation and subsequent to parturition. If the functions of secretion and excretion be checked, as by loaded bowels, or the accumulation of morbid products in the blood, a source of irritation to the nervous system at once appears; and, if *albuminuria* coexist, the *uræmic* state of the blood may tend towards actual convulsion.† Then come the physical and moral shock of labour, the emotional tension, and recoil of this crisis; and, lastly, the immediate and more remote consequences (local and general) of parturition.

The physical and moral shock is ever a varying quantity; but, in all cases, nervous exhaustion is always attendant on such an enormous outlay as is demanded during parturition, especially in those constitutionally enfeebled, reduced by the ailments of the later stages of gestation, by insomnia, or by a tedious protracted labour, or attendant hæmorrhage. The *moral* shock is regarded as an all-powerful excitant to mental ailments of this period, as illustrated in cases of illegitimacy; and authorities have constantly drawn attention to the prevalence of puerperal insanity amongst those who have borne illegitimate offspring. "Esquirol speaks of a sort of frenzy incident to unfortunate girls in giving birth, in misery and secrecy, to bastard children; a condition of mind, which it is to be feared, often prompts either infanticide or suicide."‡

Again, subsequent to labour, we have the whole uterine surface exposing the system to the perils of hæmorrhage by imperfect contraction, to retention of excretory products, to the absorption of septic agencies, metritis, phlebitis, and its attendant evils.[*] Later, still, come the evils due to imperfect depurative processes, requisite for the removal of adventitious products during the slow involution of the uterine muscle; the fatty disintegration of the giant-fibre, and its replacement by the nucleated fibre-cell, which prevailed in the nulliparous state. The products of such disintegration found copiously in the lochia, and probably as the fatty elements in the urine, and caseous elements of the early mammary secretion, will, if these secretory and excretory functions be arrested, lead up to the evils now alluded to. In fact, the whole puerperal period is one of **extreme susceptibility.**[†] The explosive condition of the nerve centres is at its height during parturition, and it is then, especially, that eccentric irritation of the cerebrum may lead to transient psychical anomalies, or to the motor discharges of general eclampsic convulsions; and it seems but a question of individual susceptibility, or intensity of the eccentric irritation, which determines the one or the other, the psychical often preceding the convulsive phenomena. Thus, at the acme of a supreme uterine effort, and especially during the passage of the head, if large, in excitable primiparæ, the intensity of the pain is often accompanied by complete, though transient, alienation, by grave reductions in consciousness, by rambling incoherent talk, by outrageous and impulsive actions—the new-born infant may be sacrificed to the mother's frenzy.[‡]

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* *Commentaries*, p. 363.

† The uræmic element in the causation of puerperal eclampsia has been called in question; see, however, the very apposite remarks by Dr. Gowers in his *Diseases of the Nervous System*, vol. ii., p. 716.

‡ Burrows' *Commentaries*, p. 364.

Illegitimacy.—I have already drawn attention to Esquirol's statement of the prevalence of insanity amongst those who have borne illegitimate children; it has also been shown by Dr. Clouston that this cause is a potent factor in Scotland—where illegitimacy abounds. He estimates that 25 per cent. of his cases occurred where the offspring were illegitimate. When we take our English asylums into consideration, the results are far different; illegitimacy is far less rife; and it appears that out of a total of sixty-six cases sixty-one were married, and the children born in legitimate wedlock, whilst in five only were the patients unmarried women. Now the proportion of single to married patients in the total admissions of 1810 cases was 35 per cent. to 50 per cent. respectively; hence the proportion of 10 per cent. of single women who suffered from puerperal insanity is exceptionally low.[§]

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[*] See "Clinical Illustrations of Puerperal Insanity," by Dr. Campbell Clark, *Lancet*, vol. ii., 1883.

[†] Burrows, *op. cit.*, p. 366.

[‡] There is, likewise, a temporary delirium sometimes accompanying difficult labours, in the fever on the secretion of milk, or the inflammation of the breasts. Burrows, *ib.*, p. 364.

[§] Dr. Macleod estimates the frequency of illegitimacy as a factor at 11·7 per cent.—*Address on Puerperal Insanity*, 1886.

Frequent in Primiparæ.—Frequent child-bearing has apparently no connection with the development of insanity; of the sixty-eight puerperal cases, twenty-two were first confinements, or a percentage of 32·3; 20·5 per cent. had had two children; 10 per cent. and 14 per cent. respectively had had three and four children; and of those who had families ranging between five and nine, 22 per cent. were also represented.

Second attacks occurred in eight cases, of whom two were primiparæ, and the remaining six had families ranging from two to nine. No case had suffered from a third seizure.

The Blood in Puerperal Insanity.—Tested by the hæmoglobinometer the amount of hæmoglobin was found below the normal, as indicated by the series of observations recorded in the following table:—In the case of *R. W. J.* it amounted to but 55 per cent., and in *C. C.* to 60 per cent. of the standard of healthy blood; in two other cases it varied between 74 per cent. and 78 per cent. The first case of the tabulated series (*M. A. M.*) in which profound anæmia had resulted from *post-partum* hæmorrhage, gave upon one occasion as low a percentage as 20, rising subsequently to 32 per cent. Both specific gravity of the blood and the amount of hæmoglobin have been lately stated to be lowered in states of mental excitement, generally with much muscular activity (*Vorster*).

AMOUNT OF HÆMOGLOBIN IN THE BLOOD IN THE SUBJECTS OF PUERPERAL INSANITY.

	Hæmoglobin. Per cent.	Red Corpuscles. Per Hæmic Unit.	White Corpuscles. Per Hæmic Unit.	Value per Corpuscle.	REMARKS.
M. A. M., (Oct. 1, '87),	20	53·6	·25	·35	Extreme waxy pallor; blood *very pale*, watery, and instantly separates into serum on withdrawal; contains many minute cells like nuclei and ill-formed corpuscles.
,, (Oct. 4, '87),	28	41·4	·06	·68	Minute fat globules in the blood and many ill-formed corpuscles.
,, (Oct. 8, '87),	24	40·8	·12	·60	Still many minute nuclear bodies; blood pale but has more consistence.
,, (Nov. 6, '87),	32	71·6	·16	·45	Red discs all contain minute glistening nuclear bodies; some tend to form dumb-bell shapes and readily split up. *Wild excitement for some days past.*
M. A. P., (Nov. 15, '87),	74	93·6	·28	·79	Profound melancholia; waxy pallor of face; compulsory feeding requisite. Many minute corpuscles in the blood, some of dumb-bell form; larger corpuscles measure 6 to 8 μ; smaller measure 5 μ
H. S. L., (Nov. 2, '87),	78	102·8	·22	·76	Considerable torpor of movement; much stupor but no cataleptic phenomena; pupils dilated; betrays but slight anæmia.
C. C., . (Dec. 2, '87),	60	80·6	·24	·75	Medium size corpuscles (5 μ); a few small nuclei (2 μ); white corpuscles measure 8 μ.
R. W. J., (Aug. 10, '89),	55	124	·18	·44	

The corpuscular *richness* (numerical) in the uncomplicated cases came near that of normal blood,* being 80·6 to 124 per hæmic unit—*i.e.*, from 4,030,000 up to 6,200,000 corpuscles per cubic millimetre; but, the corpuscular *value* estimated in hæmoglobin was invariably low—the lowest register being ·44 in the case of *R. W. J.*, the other three subjects giving ·75, ·76, and ·79 respectively of the normal value. In the case of *M. A. M.*, however, the numerical corpuscular richness, commencing at 2,680,000, fell to 2,040,000, and eventually rose to 3½ million corpuscles per millimetre cube; the corpuscular *value* at one time being ·35 rose to ·68 and subsequently fell to ·60 and even ·45—the latter coincident with a wild maniacal outburst.

Prognosis.—Of the seventy cases, fully fifty-six completely recovered at the asylum, whilst four others were discharged "relieved"—hence the *recovery-rate* reached the favourable percentage of 80. The mortality was 8·5 per cent. of the whole sixty-eight cases—*i.e.*, six died. On the other hand, four patients (5·7 per cent.) still remain in the asylum as chronic incurable cases of several years' standing. On consulting the recovery-chart it is observed that, up to the second month, but nine cases recover; thence, up to the sixth month, the recoveries rise gradually to an aggregate of thirty-seven; during the following two months, but five cases recover; at the ninth month there is a sudden rise in the recoveries, from which period, up to two years and a-half, a few casual recoveries are still noted. The recovery-rate does not, therefore, contrast so favourably as appears in Dr. Clouston's statistics, in which it is stated that in three months over half had recovered, and in nine months 90 per cent. were well. Our own results show that rather more than one-half of the recoveries occurred by the fifth month, and an advance of the number of recoveries to forty-four by the end of the sixth month; whilst, as in Dr. Clouston's cases, 87·5 per cent. had recovered by the ninth month from the commencement of their insanity. In an admirable summary by Dr. Macleod of a large number of cases of puerperal insanity collected from English and Scotch asylums, the recovery-rate is given at 77·3 per cent. of the total. His statement is as follows:—"Of 814 cases of puerperal insanity, 620 recovered, or 77·3 per cent. Of these 814 cases, 74 died from all causes, giving a mortality rate of 9 per cent." †

The incalculable advantage of early treatment is very obvious in the recovery-list; since of four-and-twenty who were placed in the asylum within one week of the onset of their insanity, as many as thirteen were recovered within three months, and the remaining eleven left recovered within five months, and this *despite the fact* that several of these patients had inherited strong neurotic tendencies; thus, the family records of these twenty-four (early) recoveries testify to the following facts :—

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* Assuming normal blood to be correctly computed at 5,000,000 per cubic millimetre.

† *Loc. cit.*

Mother paralysed, ...	Recovery in 2¼ months.
Brother insane, ...	,, 5 ,,
Mother insane, ... Sister insane; father a heavy drinker, .	,, 2½ ,,
Sister insane, ...	,, 5¼ ,,
Grandfather insane and committed suicide, .	,, 2 ,,
Father insane, ...	,, 1 ,,
Father and mother insane (second attack), .	,, 4¼ ,,
Aunt and two cousins insane (second attack),	,, 2¼ ,,
Aunt insane, ...	,, 4 ,,

Even direct inheritance does not, therefore, seem so strongly to affect the recovery-rate in such cases when placed at an early date under suitable treatment.

Let us now turn from the favourable cases to those in which recovery was protracted to six months and later. It becomes a significant fact that these are cases where asylum treatment has been deferred, and the patient kept under their friends' supervision for a period ranging from two weeks to several months.

Case.	Onset prior to Asylum Treatment.	Recovered.	Age.
1	4 weeks	7 months	26
2	2 ,, (brother insane)	7 ,,	36
3	2 ,,	7 ,,	22
4	3 ,,	7¼ ,,	25
5	4 ,,	9 ,,	24
6	3 ,,	9 ,,	24
7	?	9½ ,,	25
8	6 months	9½ ,,	48
9	7 ,,	9 ,,	38
10	2 ,,	9 ,,	21
11	3 ,,	10 ,,	24
12	3 ,,	11 ,,	27
13	2 ,,	11½ ,,	25
14	3 weeks	12 ,,	29
15	2 ,,	13½ ,,	22
16	3 months	14 ,,	27
17	2 ,,	2 years	27
18	?	2½ ,,	24
19	7 weeks	6¼ months	25
20	7 ,,	6¼ ,,	22
21	9 ,, (paternal heredity)	6¼ ,,	26
22	2 ,, (,, ,,)	6½ ,,	28
23	4 months	6 ,,	28
24	1 ,,	6 ,,	24
25	25 ,,	6½ ,,	25

Equally instructive is it to note, that the failures amongst those who were but partially relieved and so discharged, or who remained as chronic insane, or who succumbed to a fatal malady were, with very few exceptions, either *late admissions or over thirty years of age.* A glance at the following three tables illustrates this point in a very forcible manner :—

MORTALITY.

Duration of Attack on Admission.	Age of Patient.	Date and Cause of Death.
3 months,	27	in 11 months ; of tubercle.
3 ,,	38	in 6¾ years ; of phthisis.
Several months,	42	in 1½ years ; of general paralysis.
7 months,	40	in 1 month ; of pneumonia.
1 ,,	21	in 3 months ; of phthisis.
11 ,,	28	in 2 years 7 months ; of general paralysis.
1 week,	30	in 1 month ; of pelvic cellulitis.
1 ,,	36	in 2 weeks ; of chronic brain atrophy.
1 ,,	40	in 1 week ; of pulmonary congestion.
1 ,,	35	in 4 months ; acute cerebritis (father and mother insane).
10 days,	22	in 4 years ; of phthisis.

DISCHARGED "RELIEVED."

Duration of Attack on Admission.	Age of Patient.	Date of Discharge.
2 weeks,	30	2 years.
3 months,	29	6 months.
5 weeks,	39	2 years and 4 months.
7 months,	38	2 months (father and brother insane).
Several months,	32	8½ months.

REMAINING "CHRONIC INSANE."

8 weeks,	34	7 years ; still an inmate.	
6 ,,	26	7 ,, ,,	strong heredity.
8 months,	39	1 year and 8 months.	
1 week,	30	4 years.	
1 ,,	28	5 ,,	

In conclusion, then, it may be stated that the insanity occurring at the puerperal period is one of most **acute** character, yet most **favourable** as regards the ultimate issue of treatment.

The prognosis will be influenced more especially by—

(*a*) Duration of the symptoms when the patient is brought under treatment, and

(*b*) Largely by the age of the patient.

If the patient be under thirty, and judicious treatment be employed within a week of the onset, the prognosis is favourable; every day's delay after this adds to the ultimate risk.

If the patient be over thirty years of age, and, more especially, if the treatment has been delayed for two weeks or longer, then the recovery if ensured will probably be prolonged, yet the risks of *partial recovery only* will be strengthened.

Treatment.—Our first enquiries should be directed towards the genito-urinary system, with the object of discovering any local uterine mischief, whether tenderness upon palpation, a rising temperature, a history of arrested or fœtid lochia, indicate lurking troubles consequent upon labour. The most careful examination should be made with the object of ascertaining any source of peripheral excitation which may be removable or palliated. A slight pyrexial movement will generally be found in the acute maniacal attacks following immediately upon parturition. It is always advisable to begin our treatment with a saline aperient, and secure a free evacuation of the bowel; the condition of the breasts may also merit attention. A bland but nutritive and fluid or semi-fluid food (including milk, beef-tea, broths, and eggs) should then be given at very regular intervals—forcible feeding being resorted to should the exigencies of the case demand it, and, in fact, this is usually the rule. The condition of the blood would appear to indicate the administration of iron; but, in all these cases of acute excitement, it is well *not* to give chalybeate preparations until a much later date in the history of the case; at all events, not until the patient takes food spontaneously and sleeps fairly well, without having recourse to sedatives.

The form of sedative and soporific is of importance; bromide salts are of less avail singly than in combination with chloral. The latter is the better drug to rely on in obstinate insomnia. Paraldehyde has been given with considerable success, and is certainly more efficacious than sulphonal; but, of the three, we give the preference to chloral. Later on, with abatement of maniacal symptoms, it will be well to administer phosphatic foods, the syrup of the phosphates, or a mixture of the ammonio-citrate of iron along with malt extract.

In later stages, the question of uterine involution is one of much moment—undoubtedly a defective involution has much to do with the persistent excitement of these cases. Here Easton's syrup, the liquor strychniæ, or tincture of nux vomica, may be of avail; or the chloride of ammonium may be given with advantage.

Early association with the insane should be avoided, as likely to increase the irritative process going on. The subject should be kept at first confined to her bed, attended by a nurse, and only when the maniacal excitement is somewhat abated should she be taken into the open air for short walks, or allowed to associate with others similarly deranged. Warm baths may be utilised with advantage during the progress of the case, and by their aid it is possible to ensure sleep in many cases without resorting to the employment of sedatives. Macleod wisely insists upon the re-establishment of menstruation during convalescence as an important element in treatment. "No case can be considered as cured till the menstruation is regular; aloes and iron pills, ergot and warm hip-baths are indicated. I have found potassium permanganate pills most useful in this respect."

Insanity of Pregnancy.—Insanity during the period of gestation is remarkable for the infrequency of its occurrence; upon this point all authorities are agreed. Our statistics in large pauper-asylums certainly corroborate this statement, and, when we deduct the cases admitted subsequent to confinement, but whose mental ailment definitely dates from a period prior to parturition, and only estimate those who were *enceinte* on admission, we reduce the numbers so far that they are of little or no value for statistical purposes. Eleven cases of insanity occurring during gestation were admitted amongst a total of 1,814 female admissions, or the very low proportion of 0·6 per cent.; hence our experience with respect to insanity at this physiological cycle is indeed greatly restricted. So great is the repugnance to the admission of such cases, and very naturally so, that undoubtedly many subjects escape asylum supervision, and are nursed through transient attacks of alienation under the guardianship of their friends at home; and, on the other hand, so frequent are the trifling mental ailments of the earlier months of pregnancy—the morbid cravings, the emotional and moral perversions—that they are regarded with little concern; whilst a more serious mental ailment may be excused as but an exaggerated expression of the same states. Our very limited experience at the West Riding Asylum would indicate that insanity at this period is by no means more prevalent amongst *primiparæ*, for *nine* of the eleven cases had previously borne children, and in none of these cases had the patient suffered from a previous attack of insanity. Cases have been recorded where every confinement has been preceded by mental disturbance amounting to genuine insanity; but it is much more usual to find such frequent recurrence as the sequel to successive parturitions, than in the pregnant periods of a woman's life.

There is usually a period of mild depression for some time observed ere the more acute outbreak of symptoms; nervous timidity is a frequent accompaniment; the patient loses confidence in herself, and dreads that some imaginary evil is about to befall her; she becomes suspicious, and often exhibits want of confidence in her husband and relatives. All the cases which required removal to the asylum were instances of acute maniacal excitement; they were not associated with expansive emotional states but the reverse. Distrust, timidity were apparent in all; and, at times, terror induced by acute hallucinations culminated in frequent frenzied excitement and the wildest conduct. Periods of sullen reserve would alternate with sudden outbursts of mania; and the subject was usually watchful, intensely suspicious, and suddenly aggressive. There was in most of the cases a special danger of suicidal acts, attempted usually under the influence of some terrible delusion; as in the case of one patient who believed herself seized by Satan, and who made frantic efforts to leap through a window. The excitement is accompanied by great incoherence; but their ramblings usually betray the dominant feelings in frequent reference to bloodshed, murder, treachery, or the like. The most persistent insomnia often prevails, and destructive tendencies are at first obvious. Later on the patient may have alternations of depression and mild excitement; in which she is flighty, meddlesome, treacherous, and prone to vicious conduct.

The larger proportion of cases occurred *beyond the third month* of gestation, and two cases alone left the asylum recovered before their confinement. The proportion of "recoveries" amounted to 54·5 per cent., whilst two others left, after a prolonged residence, sufficiently relieved for home treatment; two, however, died—one from puerperal fever, and the other of chronic phthisis.

With respect to the origin of the mental derangement we can predicate but little from the scanty figures at our disposal and the history of the few instances afforded us. It was ascertained, however, that strong *hereditary predisposition* prevailed in 36 per cent.; that *two* other cases subsequently died of *general paralysis;* and that *three* others, although affording no history of ancestral insanity, were considered to be of *congenitally-defective mental organisation.*

INSANITY AT THE PERIOD OF LACTATION.

Contents.—Risks Attendant upon Lactation—Period of Uterine Involution—Period of Mammary Excitation—Symptoms—Depressing Delusions—Impulsive Nature (M. W.)—Suicide (M. D.)—Intensity of Maniacal Excitement—Sexual Perversions—Hallucinations—Etiology -Exhaustion and the Sequelæ of Labour—Protracted Uterine Involution—Lactation during Profound Anæmia—Hyperlactation—Qualification of the Nursing Mother—Period for Weaning—Prognosis—Treatment.

To a certain proportion of the puerperal, the whole period of lactation is one fraught with risks. The period is one of acknowledged susceptibility, and when conjoined to this normal exaltation we have the predisposition engendered by ancestral insanity, the acquired elements evolved out of vicious modes of life, and inattention to the plainest physiological dicta, the morbid impetus towards insanity is greatly strengthened. Undoubtedly, the factor peculiar to this period of lactation, to which are attributable in part the mental reductions, vary with the physiological changes incident to this period. Thus, in the early period of lactation, the immediate effects of gestation and parturition, or the changes normally aroused in the uterus on the completion of labour, are of paramount importance in our estimate of the origin of the mental ailment; and so, throughout the period of uterine involution, the reflex irritation from the ovario-uterine apparatus is of primary importance. As, however, uterine involution becomes complete, so the activity of the mammary secretion assumes an increasing importance in its effects upon the economy; in lieu of *reflex excitations* from the uterine surface, or of the faults arising from defective depuration, the *nutrition of the nerve-centres* becomes more *directly* involved.

To attempt, however, to distinguish betwixt the insanity incident to these periods as distinct nosological entities would be highly inconsistent, and not justified by a scientific estimate of the relative value of symptomatic indications. The insanity of the parturient and early puerperal stage imperceptibly glides into that which characterises the later stages of lactation; and none but an arbitrary division can be assigned (for convenience in study) as the termination of uterine involution. Then, again, the completion of involution is an uncertain period, variously assigned by different authorities. One month is given as the term in healthy subjects, under good hygienic surroundings; six weeks is the accepted time in Continental Lying-in Hospitals (*Barnes*[*]); whilst Tylor Smith[†] quotes *two or even three* months as the probable period. We may, however, accept six weeks after parturition as the period when the uterus and ovaries are passing into the quiescent stage, during which lactation assumes its own important *rôle*. It is highly essential that we should clearly recognise this overlapping of physiological stages, as thereby are explained certain exceptional cases which occur about the transition-period here alluded to.

Symptoms.—The prevailing condition at this period is, in fact, that of an *acute psychosis*, in which excitement predominates, and in which terrifying hallucinations (visual and aural, or combinations of these) lead up to various delusional notions, and in which suspicion of friends, relatives, and neighbours is prominent; fears of supernatural agency are not infrequent, strange phantasms haunt the eye, mysterious whisperings, unexplained sounds, or more definite voices issue in the morbid imagery of angels or ministers of darkness. The loss of self-confidence begets the frequent notion that the "soul is lost," or that all future good is sacrificed by some imagined crimes committed. The moral being has its ultimate foundations shaken, and confidence is lost even in those who should be nearest and dearest to the afflicted one; the husband's fidelity is openly challenged; intrigues of acquaintances dreaded; the food declared to be poisoned.

[*] *Diseases of Women*, p. 469.

[†] *Manual of Obstetrics*, p. 92.

The **onset** may be sudden; it is far more frequently preceded by mild depression. The patient becomes restless, irritable, variable in mood, suspicious of her friends, impatient and fretful; she is apt to misinterpret the conduct, gestures, and words of others. Then come fitful outbursts of anger, extravagant accusations, or actual violence, and the onset of genuine maniacal symptoms. Insomnia usually prevails; noisy, boisterous, incoherent ramblings ensue, in which the patient gives utterance to fragmentary sentences from which we glean the condition of mind to be one of distrust, suspicion, or terror, or to be dominated by aural hallucinations.

The **delusional notions** vary from time to time, and periods of exaltation alternate with mental pain, rising even to the pitch of acute melancholia. Such subjects usually come under our notice in asylums, looking exceedingly pallid from anæmia, reduced, thin, and jaded from continued sleeplessness and excitement. In this stage of excitement they are often dangerously **impulsive,** and require most careful watching. Thus, one of our patients, who had suckled her infant up to the twelfth month, although much enfeebled in health, became suddenly maniacal at home; she struck her husband on the head with a poker, ran a darning-needle into his side, and eventually got possession of a knife at night and gashed his throat ere she could be secured.

M. W., aged thirty-nine, married, and the mother of three children. Patient inherited insanity from the mother, who was an inmate of this asylum twenty years ago after confinement. The patient was confined twelve months prior to admission, and had brought the infant up at the breast until four months since, when depression first supervened: her health began to fail, restlessness, low spirits, suicidal feelings, and the delusions that her soul was lost, that the devil was in her house, and that all her friends had become her enemies, characterised this period. She had suffered from continued insomnia. Patient had been of temperate habits.

She was communicative upon admission, and discoursed readily upon the subject of her mental ailments. Ever since weaning the child she had been depressed, filled with morbid fancies, frequently felt tempted to injure herself, had lost all control, and (unless closely observed) would certainly have destroyed herself. Had found her memory much impaired of late, and this had troubled her greatly; she had also taken a deep-rooted dislike to her home, because she fancied that a friend who lived opposite was constantly watching her. Admitted that she *believed her soul to be lost, and felt given-up to despair.* The catamenia had been regular and normal for some months. Cod-liver oil emulsion ordered, and a mixture containing 10 min. of liq. opii, and aromatic sulphuric acid three times daily.

From this date she made a steady progressive improvement, although restless at nights, and suspicious of the patients with whom she was associated; she was much given to brooding and introspection, but recovered sufficiently to employ herself in household occupations within two months of her admission to the asylum. Then came a relapse with *sudden suicidal impulse*, and it is noted, November 2nd—"This morning, when employed in the chief female officer's bedroom, she was caught in the act of *suspending herself to the bed-post by the blind-cord.*"

She was again placed under opium treatment, which had been discontinued for a time, and from this date onwards physical and mental improvement continued, and she was discharged recovered, after a residence of eight and a-half months.

Another young girl in the sixth month of nursing was admitted in a state of rambling incoherence, flighty, erratic, and given to silly laughter; reduced in health, and anæmic, with a hæmic bruit. She violently assaulted her mother, and nearly succeeded in throttling her father, whose identity she appears to have mistaken.

Others make equally determined attempts upon their own life, as in the case of a poor weakly woman, who, struggling against the odds of penury, had been suckling *other infants* besides her own for a period of twelve months with barely a subsistence diet. Upon admission she made a most desperate attempt to drown herself in a bath in which she was placed, fiercely struggling with the nurse in her efforts to keep her head under water.

M. D., aged thirty-two, married, and the mother of three children, showed her first symptoms of mental alienation four weeks prior to admission. Four months before this date she gave birth to twins, and had suckled both up to the present time. The history testifies to great depression, with attacks of intense and prolonged excitement, with incoherent raving, and violent aggressive conduct. She had frequently threatened to take her own life, and had once tried to cut her throat. The family history was defective, but an aunt is stated to have died in an asylum.

On admission (Oct. 18th) she was low, despondent, and emotional; querulous and discontented. She conversed readily upon the subject of her depression, and narrated how she had felt low and depressed, even back to the early months of pregnancy, when she used to visit a neighbour's farm, where there was a great black dog; when she became more despondent she developed the delusion that her child would be like a dog. She was at this time very thin and delicate; the complexion anæmic and blanched, the eyes sunken with dark pigmented areolæ around. The breasts were swollen, distended with secretion, and tender. Oleum ricini was at once administered, and belladonna liniment applied to the breasts. The patient was ordered full extra diet and chloral, if necessary, at night.

A week later, she was greatly depressed; stated that she had suffered when at home from *impulses to kill her children*, but denied this fact to the doctor. She now regards this artifice as a great sin for which she cannot be forgiven.

Nov. 1st.—Is most determinedly suicidal, and sleepless at night. Last night she tore up her sheet and attempted strangulation, and this morning repeated the attempt with her apron-string. Expresses her determination to die. Tinct. opii., mins. xxx, ter die.

Nov. 27th.—To-day attempted to swallow a needle, which became impacted in the pharynx, and was removed with some difficulty.

Nov. 29th.—It was discovered by the appearance of swelling and stiffness of the neck, that she had also on the 27th inserted a darning-needle there; she was questioned on the point, and admitted the act. There was no mark in the skin, but by pressure on the opposite side of the neck a prominence could be felt over the left sterno-mastoid muscle, just in front of the external jugular. It was cut down upon, and a large darning-needle removed.

Dec. 14th.—After slight improvement has again become desperately suicidal;

tries to choke herself by anything she can lay hands upon, and constantly seeks to obtain needles by stealth.

Dec. 16*th.*—This morning a sharp projection like the head of a needle was felt beneath the skin on the right side of the neck, about the middle of the sterno-mastoid. An unsuccessful attempt at removal was made, owing to her desperate struggling to prevent it. On the 26th inst., the prominence was again felt, and a needle an inch and a-half in length removed. She is still greatly depressed and suicidal.

Then follows an account of *peritonitis*, during the course of which no foreign body was detected by external palpation ; but the patient rapidly succumbed to the attack, and died on the 14th of January. A *post-mortem* examination exhibited a localised peritonitis, the coils of small intestine being matted together by a considerable amount of lymph and purulent material: the large bowel had escaped implication. Slight ulcerative points, with patches of intense congestion, were revealed along the jejunum and duodenum, behind which was a purulent collection in which a needle 1½ inch long lay embedded. Three other needles were found in the substance of the mesentery, and one in the tissues of the neck ; none were present in the stomach or intestine. In the stomach was a piece of charred wood, 3½ inches in length by about 1 in thickness.

A similarly enfeebled, exsanguine, nursing mother, reduced by seven former pregnancies and nursings during the period of ten years—the first accompanied by puerperal convulsions, and each subsequent puerperum followed by severe headache and symptoms of exhaustion—still persists in nursing, despite four months' warning of steady progressive enfeeblement. In a state of acute melancholia so induced, she rushed off one morning with the intention of throwing herself into the river Nidd, but was arrested in the act by the "sudden remembrance of her child at home ;" she returned home, and next day swallowed a large quantity of laudanum.*

The character of the maniacal state is especially one of intensity. It is essentially an *acute mania* with or without hallucinations; but, yet, it is distinguished from the still more intense excitement of the early puerperal alienation, just as ordinary acute mania is distinguished from the furor of epilepsy. The *puerperal* form is peculiarly prone to wild, impulsive, indiscriminate conduct, as the outcome of very extreme reductions; it is peculiarly a convulsive affection, as in epileptic furor; but such profound reductions do not present themselves in the mania of early lactation. Occasionally, but very rarely, acute delirious mania may occur, as in the following case :—

S. M., aged thirty-one, married twelve months ago, and delivered of her first child three months before her admission. The labour had been natural, but her health, previously reduced, had become progressively worse, and she had been wholly incapable of attending to any household duties. Still she nursed her infant at the breast, and persisted in doing so until four days ago, when sudden and intense maniacal excitement supervened. No predisposition was discoverable, and the family history was avowed to be free from insanity, epilepsy, or apoplexy. The parents were both living and healthy. There was no moral element involved in the causation. On admission she was found considerably reduced, pale, feeble, tremulous; the pupils widely dilated; there was great anæmia; the breasts were considerably distended. She was in a state of the wildest excitement, absolutely incoherent, and utterly oblivious to the nature of her surroundings. No rational or even coherent reply could be obtained from her, but she occasionally repeated a word she had heard uttered just before. She was almost incessantly restless, and could with difficulty be kept in bed. At home she had obtained no sleep, and persistently refused food since her attack. Abundant strong nourishment, with extract of beef, was ordered, and half-drachm doses of chloral to be given at bed-time.

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* The *suicidal propensity* was witnessed in 31·8 per cent. of our cases; *impulsive violence to others* in 59 per cent. The subjects of puerperal insanity gave the lower estimate of 26 per cent. and 47 per cent. respectively.

Fortunately, she took her food readily, and the first draught procured her a little sleep. For four days she continued in a state of acute delirious mania, quite incoherent, and extremely prostrate, the tongue dry and coated, the lips covered with sordes. She then became somewhat calmer, and in six days was able to sit up in the day-room, being fairly quiet and manageable. It was not until six months had elapsed from her admission that the catamenia were re-established, after which the patient's mental symptoms (which consisted of very slight excitement, suspicion, and unreasonable irritability) passed off entirely, and in a few weeks she left recovered.

The intensity of excitement is accompanied in these cases by great incoherence and much motor agitation; but, in many instances, the patient is dominated by delusions, is reticent, evasive, suspicious, yet watchful, and (as already affirmed) dangerously impulsive.

The delusional conceptions are strikingly similar to those found in the early puerperal weeks, when, as we have seen, the idea of eternal punishment of the lost soul, of ruin and misfortune to self and family, of persecution at the hands of husband or neighbours, are the more prevalent perversions; the idea of poisoning is less frequently expressed in the insanity of the puerperal months than at this period.

In a small minority of these forms of alienation, exalted notions are apparent. They are usually evolved out of religious conceptions, the patient falls into ecstatic states, clasps her hands and is wrapt in prayer, or, maybe, believes herself to be Christ. With such religious delusions, however, far more frequently prevail obscure notions of demoniacal agency; the black-art or witchcraft is by no means an unfrequent form of explanation given. Thus, one of our patients believed herself bewitched, and called herself the "scarlet woman of Revelation;" another believed herself and husband were bewitched by the sorceries of her neighbours, whom she constantly saw peeping at her through the windows, and whose voices as constantly intimidated her; and yet another accuses her neighbours of entering her room, "crossing her furniture, and so putting all things wrong."

The **sexual condition,** if not apparent in a directly-expressed delusion, is often manifest in the patient's behaviour, indelicacy, obscene erotic language or gesture; but sexual delusions and hallucinations are by no means infrequent. The revulsion to husband and children often prompts to violence; one poor woman whose case we recall, publicly disowned her child, and then attempted to smother it. Of sixty-six cases recorded of "lactational insanity," forty-six—*i.e.*, 69·6 per cent.—had delusions.

Hallucinations of the special senses were expressed in twenty-two (33·8 per cent.) of our cases; and, hence, were of more frequent occurrence than in ordinary *puerperal* insanity. Visual and aural may occur separately or conjointly; but the prevalence of the *aural* was notably greater and more pronounced, even when both senses were affected. They were always of a depressing and painful nature; and many of the delusional notions referred to were based thereupon.

The vivid nature of such creations, the enfeebled frame of the patient, the complete loss of self-assurance, and the resulting anguish induced, produce a picture which strongly enlists our sympathies, and is painful to witness. Thus one of our patients is surrounded by spiritual beings—angels—fiends—who tell her that her soul is damned; another is tortured by abusive epithets continually shouted to her down the chimney; another hears the baying of furious dogs, and sees her children killed before her eyes. In their intrinsic nature, therefore, these hallucinations are similar to those of puerperal insanity.

Upon analysing the various forms of alienation from which our sixty-six cases suffered, we find them, as contrasted with the puerperal cases, distributed as follows:—

Forms of Mental Ailment.	Lactational.	Puerperal.
Simple mania, ...	6	7
Acute ,, ...	18	31
Acute delirious mania, ...	1	...
Mania with prominent delusions, ...	11	7
Recurrent mania, ...	3	...
Dementia with excitement, ...	1	...
Simple melancholia, ...	3	8
Acute ,, ...	3	1
Melancholia with prominent delusions, . . .	17	14
Melancholy with stupor, ...	...	1
General paralysis, ...	2	...
Congenital mental defect, ...	1	...
	66	69

It is clearly apparent, then, that at both periods excitement predominates over depression, and a larger proportion of acute maniacal attacks characterise the puerperal period. On the other hand, acute depression (melancholia agitans) is rarely met with, whilst melancholy with delusions is frequent at both periods.

Excitement in the Early Months.—The more acute forms of excitement prevail within the *first three months following parturition*; and the delusions of persecution and associated gloom and despondency of melancholia, predominate where mental symptoms first betrayed themselves *six months or more after labour.* Thus, of twenty-seven cases where alienation occurred within the first six months, there were eight cases of melancholia to seventeen of mania; whereas, in twenty-seven cases, between six and twelve months subsequent to labour, there were ten of depression to fourteen of excitement; and, later still—after twelve months—there were but eleven cases, viz., seven of depression and four of maniacal excitement.

Etiology.—It is highly conducive to the correct appreciation of these forms of mental disturbance that we keep in view the numerous factors which may operate as exciting causes of the attack; we are *not dealing* in the majority of our cases with a *simple agency*, such as hyperlactation; we have to consider not only the peculiar mental temperament and physique of the nursing mother, but also the series of accidents which may have occurred before, at, or subsequent to labour, and their often far-reaching results; we have to bear in mind the possible divergence from normal physiological reversions of the ovario-uterine system, as well as the prolongation of suckling beyond the limits which the mother's health will stand, the deprivation of food which penury may entail, and the long list of moral agencies which poverty and wretchedness so frequently call up. Amongst the more important of this category of causes are :—

1. Severe protracted labour with instrumental delivery.
2. Serious *post-partum* hæmorrhage, or the flooding occurring before or during labour.
3. Protracted or arrested uterine involution.
4. Lactation where profound anæmia already exists.
5. Lactation continued after the appearance of genuine mental alienation.
6. Hyperlactation.

Exhaustion and Sequelæ of Labour.—As regards the first two causes (which give rise more frequently to mental alienation at an earlier period)—the so-called puerperal insanity—it is but what we should expect that the attendant exhaustion or anæmia, if it does not at once issue in mental derangement, will so result if the tax of lactation be severe; and as illustrating this we append the following from the prior history of a few cases of insanity during lactation occurring over six weeks *after* labour :—

Case 1. Primipara, labour very protracted and severe.
,, 2. Instrumental delivery, severe *post-partum* hæmorrhage.
,, 3. Protracted labour, profuse hæmorrhage.
,, 4. Instrumental delivery, profuse flooding, phlegmasia.
,, 5. Very severe *post-partum* hæmorrhage.

Case 6. Severe flooding during gestation, menorrhagia at all catamenial periods, profound anæmia.
,, 7. Menorrhagia at all monthly periods ; profound anæmia.

In the foregoing series, it will be observed, one case alone was that of a primiparous subject; and, in fact, insanity during lactation is infrequent after first confinements. In the insanity occurring at, or shortly following, labour (*puerperal insanity*), primiparæ play a far more important part; 32·3 per cent. of such cases being first confinements. The insanity occurring during lactation presents us with but 16·6 per cent. as primiparous subjects.*

Uterine Involution.—We have strong reasons for suspecting that defective uterine involution plays a most important *rôle* in the earlier cases of lactational insanity; and it is to this cause we may often attribute the preponderance of excitement over depression in the earlier months of lactation. The *reflex* nature of the neurosis closely approximates to that which we know to be largely dependent upon the direct agency of a gravid or parturient uterus; and the results of treatment, moreover, directed towards this condition, warrant us in assuming protracted involution as being a large factor in these earlier weeks of lactation.

Suckling in a stage of cerebral exhaustion is, perhaps, of all causes the source of the more severe and prolonged insanity at this period. It is by no means uncommon to find mothers suckling their infants several months after the appearance of indubitable indications of profound cerebral derangement, loss of memory and attention, morbid feelings and desires, hallucinations of sight and especially of hearing, complete change of disposition—yet the infatuated patient clings to the habit, and will say, as one of our poor patients did to her medical attendant—"I cannot give up the child, but it will drive me mad." We need only glance at the accompanying table, abstracted from the history of sixty-five cases of insanity during lactation, to have enforced on our minds the importance, as an etiological factor, of such vicious persistence despite failing vigour, progressive anæmia, and even well-marked mental disturbance.

Case.
1. Suckled infant nine months, and for two or three weeks indubitably insane.
2. ,, infant seven months, and for several weeks had been failing in health and ailing mentally.
3. ,, several other infants, as well as her own, during the year.
4. ,, infant nine months, whilst extremely delicate and in feeble health.
5. ,, child for four years, and had always indulged in prolonged lactation.
6. ,, infant four months, although for years depressed.
7. ,, infant fifteen months, last three months during alienation.

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* First labours, 16·6 per cent. Third labours, 22·7 per cent.
Second ,, 13·6 ,, Fourth ,, 19·6 ,,

Case.
8. Suckled infant eleven months, yet peculiar and deranged throughout the whole period of lactation.
9. ,, infant nine months, yet for three months distinctly deranged in mind.
10. ,, infant ten months; several uræmic convulsions three months after confinement.
11. ,, infant four months, yet has been insane for two years.
12. ,, infant ten months; in very feeble health and deranged of late.
13. ,, infant, whilst becoming for four months past steadily and progressively enfeebled and blanched from anæmia.
14. ,, infant nine months and a-half, although for three months deranged.
15. ,, infant nine months, yet depressed for years.
16. ,, twins four months, yet for one month deranged in mind.

Injudicious nursing, therefore, plays a very important part in the causation of many of these cases of insanity; but in all such cases alike, it is to be noted that cerebral malnutrition from anæmia, whether induced by hyperlactation, by copious hæmorrhages, by the cachexy of intercurrent fevers, by phthisis, or causes associated with semi-starvation, arouses the self-same form of mental anomaly.

To supply the wants of the infant with a food sufficient in quantity and of proper quality, the nutritive processes of the mother and tissue-metabolism must maintain a well-regulated balance. The mammary secretion requires not only saccharine and fatty, but nitrogenous materials in certain due proportions, and this demand is made upon the blood-current of the gland. The relative amount of circulating albumen and carbo-hydrates in the blood is apt to be powerfully affected by many agencies, apart from the actual amount introduced by the food. Should the respiratory function be diminished or retarded, as by living in badly-ventilated, overheated rooms, the carbo-hydrates must accumulate in the blood current, and tend to be deposited more largely in the tissue; the same result must accrue in anæmic states where the oxygen-carriers—the red corpuscles—lessened in numbers, fail to oxidise the hydrocarbons circulating in the blood. In the nursing-mother the accumulation of hydrocarbon thus brought about must issue, more or less, in a relatively-increased amount of fatty matters in the milk (as, for instance, occurs in stall-fed animals). On the other hand, should frequent exposure to cold stimulate the respiratory functions and oxygenation proceed more vigorously, the hydrocarbons are diminished and the protein-compounds undergo a relative increase, so that the milk contains more casein, whilst the fats decrease. If much muscular exertion be demanded the protein metabolism increases and appears directly to affect the composition of the milk, increasing its casein.* The same result occurs in anæmia; it is found that the excretion of

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* Thus we are told that the cattle exposed to cold and much muscular exertion in Switzerland yield a very small quantity of butter, but an unusually large proportion of cheese.—*Carpenter's Physiology*, p. 613.

urea is increased, and *carbonic acid* diminished; in other words, protein metabolism is increased and fat metabolism lessened. It is, therefore, obvious that a due proportion of nitrogenous and hydrocarbonaceous constituents in the blood, regulated by a rational diet and by the normal metabolic changes occurring in the tissues, is an all-important feature in healthy lactation; yet the function itself may be profoundly disturbed by a vicious system of suckling. Thus frequent application of the infant to the breast not only stimulates the secretion of milk, but also modifies its quality. The quantity is largely augmented, and the milk becomes much richer in casein; hence a serious drain from the albuminous constituents of the blood is occasioned and anæmia results. In the nursing-mother an accumulation of storage-fat appears to occur, reminding one of the condition of hybernating animals, in which, during a prolonged winter's sleep, the entire absence of food requires (despite the cessation of activity and the extremely lowered respiratory and heat processes) a large storage of fat to supply the slow, though continuous, waste. So in the nurse, the waste occurring through the mammary secretion appears to demand a fat-storage both for the maintenance of such a secretion and the respiratory functions; the early months of lactation are consequently marked by the plumpness of the body generally—healthy nursing mothers almost invariably "put on fat" at this time of life; and it is only late on in lactation, when the system begins to appreciate a serious drain, that the rotundity of the figure lessens (*Trousseau*). It is a condition of serious moment when we find the nurse not so prepared for her arduous functions, and it is a notable fact that, in a very large proportion of cases of insanity occurring during lactation, this symptom of ominous significance is recorded. It should always be accepted as a note of warning in those who are predisposed to insanity, or who have afforded evidence of cerebral malnutrition. Such failings are usually due to injudicious nursing; and injudicious nursing is at the root of most of the cases of aberration occurring during lactation. It is not so much *prolonged lactation* as over-indulgence, often of a strong, robust infant, whilst the nurse is in a delicate state of health. If we consult our statistics we find that, out of sixty-five cases, twenty-seven had suckled but *six months*, and twenty-seven others had suckled up to the ninth and twelfth months; obviously, then, *protracted* suckling had not so much to do with the cause as other factors.

What occurs is this, a woman in delicate health, often after numerous rapidly-succeeding confinements, or perhaps after a tedious puerperum, or, maybe, hæmorrhage before or during labour, suckles her infant with injudicious frequency, sacrifices her rest night after night, and probably takes insufficient or unsuitable nourishment. The constant drain from her system and loss of rest engender anæmic and dyspeptic symptoms, still less favouring the restoration of the material lost from her blood. The superficial fat disappears to supply the demands of the exhaustive secretion. The carbohydrates, rapidly diminished, no longer can exert that protective influence over protein metabolism (*Landois and Stirling* *), and a further waste occurs in this direction—first, the circulating albumen feels the drain, and later, the organised albumen must still further aid in the formation of the fatty principles required.† In this way, as in all cases where a similar drain upon the system occurs (leucorrhœa, diarrhœa, profuse suppuration), anæmia of profound character supervenes; our patient comes before us with pallid face, with blanched lips, with small feeble pulse, the heart's muscle exhausted, and the breathing often hurried and panting, and the muscles of the limbs flabby and ill-nourished; she complains of headache, vertigo, dimness of vision, lassitude and aching limbs. In lieu of the fresh-coloured healthy glow of the cheeks, the plumpness and firmness of tissue, indicative of active functionising and vigorous health, the normal ebb and flow of lively emotion, and warm interest in the nursling, which characterise the good efficient nurse, we have a miserably pallid wasted object, half-starved, querulous, full of imaginary ailments, tormenting fears, and morbid suspicions.

Despite all these symptoms, some women will still persist in suckling their offspring from misguided maternal instincts, and often with the object of deferring a subsequent conception; the hydræmic state of the blood results in a slow and sluggish circulation, and in the splenic pulp, and especially the portal circulation of the liver,‡ as in the marrow of bone, such sluggish flow favours the destructive hæmolytic action which goes on here—the red corpuscles are rapidly disintegrated (*Quincke*).

Although it has been demonstrated that in inanition, the central nervous-system loses in weight more slowly than almost all tissues and organs (with the exception of the spleen, kidneys, and heart), and incomparably less than the fat, muscles, and even bones (see V. Voit's experiments quoted by Landois, p. 514), yet, the brain is undoubtedly the organ which earliest registers any disturbance in its nutrition. This is what we might expect; since, being the custodian of all organic impressions, to which is relegated the function of giving the alarm when the activity of other organs is disturbed, it is highly necessary that its own welfare when threatened should be expressed with no uncertain cry.

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* It must be remembered that only part of the fat is derived from the food directly; the rest is a product of a splitting-up of proteids in tissue metabolism (Landois and Stirling's *Human Physiology*, p. 518).

† Then, also, we must remember that (as Voit shows), although in ordinary health a large amount of circulating albumen is split-up, whilst the organic albumen of the organs and tissues continues comparatively stable; yet, in certain pathological states, the organic albumen becoming very unstable may undergo rapid disintegration, as in fevers, &c. (Stirling, *op. cit.*, vol. i., p. 506.)

‡ The liver is regarded as one of the chief sites of hæmolysis "because bile-pigments are formed from hæmoglobin, and the blood of the hepatic vein contains fewer red corpuscles than the blood of the portal vein" (Stirling, *op. cit.*, vol. i., p. 17).

What is the proper time for weaning? The period is, of course, a most variable and uncertain time; each individual case must be judged upon its own merits, and in considering, as we are compelled to do, the interests of both parties, the mother and offspring, the question necessarily becomes peculiarly trying and delicate, owing to the fact that the immediate interest of the one often appears antagonistic to that of the other. Ultimately we know that, if the nurse suckle beyond her strength, the result will prove highly prejudicial to both, yet, the infant's health may be such that artificial feeding is inadmissible and urgently demands breast-milk of a quantity and quality beyond the mother's capability to supply. Undoubtedly, natural feeding at the breast should be adopted in all cases where the health of the nurse is not seriously imperilled, and when the breasts supply a due amount of nourishment for the infant's wants; as, hereby, the well-being of both is best secured; but, when the lacteal secretion is scanty, or its quality such as to render it unsuitable in the case of a weakly infant, to whom artificial feeding might prove a greater peril, the balancing of odds is often a difficult matter. Consistently with the mother's health, every effort must be made to bring up the child at the breast, over the more eventful dentition crisis through which it has to pass, and this might be oftener done by weakly nurses if due regard were paid to a judicious course of suckling and avoidance of errors so notoriously frequent.

The child has to be educated into a regular system of feeding; and this should be so managed, that the mother may secure a prolonged rest and sleep at night, and the injurious habit avoided of indulging at night every passionate outcry by application to the breast.

The rule given by Trousseau holds good for most cases where we deal with a healthy nurse—viz., that the period of lactation be extended in most cases over the period of evolution of the canine teeth.

> "My rule, provided there be no serious obstacles to surmount, other than the wishes of the family, is not to wean the child until after the complete evolution of the canine teeth, which is generally a more difficult process than the evolution of the incisors or first molars. My rule, therefore, is to wait, irrespective of age, till the infant has sixteen teeth." *

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* *Trousseau, op. cit.*, vol. iv., p. 163.

Of course any such rule falls short in its application to cases such as we are now considering. The welfare of the mother, if of neurotic inheritance, and exhibiting emaciation and anæmia in the early months of lactation, is so greatly threatened as to demand the immediate cessation of nursing.

Prognosis.—The recovery-rate appears to be directly affected by the date subsequent to parturition at which the insanity appears; the *earlier* the symptoms of mental alienation occur, the *more favourable* is the issue likely to be. Hence, the recovery-rate is higher for puerperal cases—that is, cases arising within six weeks of confinement, than during later lactation, as 80 per cent. is to 65·6 per cent. The percentage of recoveries for the whole number of female cases of insanity was 44·6, so that the percentage of 65·6 represents a very favourable rate of recovery, and justifies a good prognosis, although of a less favourable nature than in puerperal insanity. (*Chart C.*)

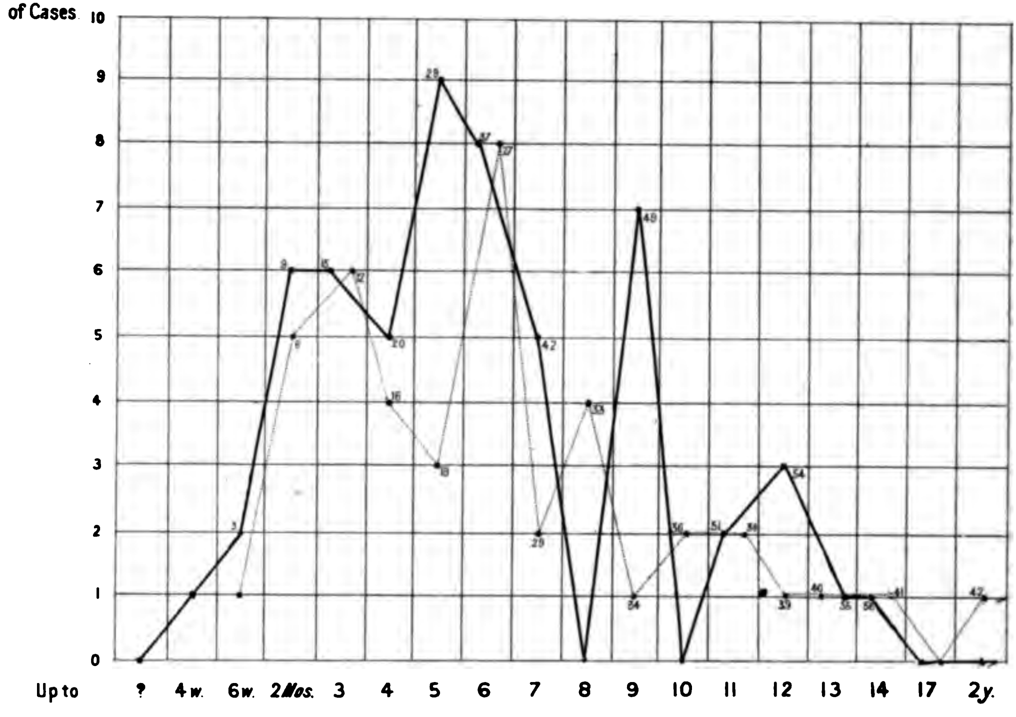
If a chart of recoveries in puerperal and lactational forms be contrasted, a notable uniformity is recognised throughout, the largest number of recoveries in both occurring at the fifth or sixth months and rapidly declining in the subsequent period. At the ninth month of puerperal females, however, a remarkable rise again occurs in the recoveries of seven cases, somewhat paralleled by a less conspicuous rise at the eighth month in lactational cases. Whatever be the cause of these wave-like recurrence of cases, it is obvious that the periods at which the recoveries chiefly tend to occur in both series of cases are from two to three, from five to six, and, again, from eight to nine months from the onset of the disease. Age exercises less apparent influence upon the number of occurring cases than upon the *nature of the attack*, and so indirectly affects the issue. The influence of age upon insanity occurring during suckling, in a series of sixty-five cases where the age was definitely given, may be illustrated by the following table:—

Age.	Recovered.	Relieved.	Died.	Remained Chronic.	Total.
From 15 to 20	1 (at 19)	...	...	...	1
,, 20 to 25	9	2	2	...	13
,, 25 to 30	15	3	1	2	21
,, 30 to 35	13	1	3	3	20
,, 35 to 40	5	1	2	1	9
,, 40 to 45	...	...	1 (*G. P.*)	...	1
	—	—	—	—	—
	43	7	9	6	65

If we compute as failures the fatal and chronic cases, it will be observed that the ratio of these unfavourable cases steadily augments towards the age of forty, increasing from one-sixth of the whole at the age of twenty, to one-third at the age of forty. The large proportion of cases of insanity appear from the total column in the above table to

CHART (

CHART of RECOVERIES in 70 PUERPERAL & 67 LACTATION CASES.



NOTE.- *Black Line indicates the* PUERPERAL
Dotted Line . . LACTATIONAL FORMS.

ANALYSIS OF RESULTS.

	Recovered	Relieved	Died	Chronic
Puerperal	56 or 80%	4 or 5·7%	6 or 8·5%	4 or 5·7%
Lactational	44 or 65·6%	6 or 9%	8 or 12%	9 or 13·4%

BALE & SONS & DANIEL

have occurred between the ages of twenty-five and thirty-five. We find, moreover, that the proportion of maniacal to depressed, melancholic subjects is far greater at the earlier age of twenty to twenty-five, and progressively lessens until, at forty, excitement and depression prevail in about the same relative frequency. It would thus appear, at first sight, that youth is favoured as regards greater immunity from such affections; in the attack being more acute, and hence more favourable; and in the ultimate issue in recovery. This would not, however, express the case truthfully, since the proportion of married women in the population of our asylum-district between the ages of twenty and twenty-five is considerably below the married population of the following decennial period of life. In fact, between the ages of twenty-five and thirty-five, the population is nearly 122,000 as against 37,560 who are from twenty to twenty-five years of age; this represents a ratio of 3·246 to 1. Taking, therefore, into consideration this fact, that the number of married women in the West Riding between the ages of twenty-five and thirty-five is *more than triple* that of the married from twenty to twenty-five years of age, it is seen that the younger class do *not* share an immunity from the ills attendant on lactation; the incidence of insanity at these ages being almost identical. That depression prevails in the later quinquennials and that unfavourable results multiply disproportionately may be regarded as established, so far as these statistics lead us to infer. A fatal issue in these cases of insanity during lactation is not to be feared apart from the complication with other affections, especially the phthisical tendency, which is prone to declare itself at this epoch; more than half the deaths were due to this cause in our own statistics, two others issued in general paralysis, and the remaining fatal case was one of suicide, already recorded.

Treatment.—In most instances our patient's bodily condition claims the chief attention; her strength must be nursed and supported by every possible measure. Should the breasts be tumid, we must treat that condition by the employment of belladonna inunctions, by atropine internally, by local friction, and by gentle saline laxatives. A general tonic regimen must be from the first enforced. The food should be liberal, nutritive, and easy of digestion; for errors, digestive and assimilative, are almost of constant occurrence in such cases. The peptonised and pancreatised preparations, and zymine in intestinal derangement, may be utilised with advantage, more especially if we endeavour to associate therewith cod-liver oil and malt extractives.

Combinations of iron with arsenic, or with other mineral nervine tonics, are especially useful, and the physical improvement so induced is usually accompanied by a decided mental reinstatement—depression abates, and a healthier tone prevails. The sulphate of iron, in combination with aloetic purgatives as a pill, is a useful remedy for the constipation associated with the hepatic and intestinal torpor of some of these cases; cascara sagrada may also be given here with advantage alone, or, better still, in combination with euonymin. Shower-baths, cold spinal douches, and open-air exercise are applicable in most instances. It is not, as a rule, advisable to adopt sedative treatment, except in the more intense forms of depression, when morphia given subcutaneously, or the liquor opii, will prove the more useful drugs; we should trust far more to general hygienic means for restoring tone to the system, improving the appetite, and the digestive and assimilative powers, and for inducing sleep.

INSANITY AT THE CLIMACTERIC EPOCH.

Contents.—Symptoms—A Subacute Delusional Melancholia—Suicidal Tendency (S. H.)—Nymphomania (A. A.)—Etiology—Incidence of Insanity at different ages in 4085 cases—Influence of the Climacteric—The Psychological Transformations of this Epoch—Instinctive Actions—The "*Time-element*" in Prognosis—Alcoholism and the Climacteric—Treatment.

Symptoms.—The mental ailment most frequent at this period in women is an affective insanity, in which gloom and despondency are associated with paralysed energies, indecision, and volitional inactivity; a condition pertaining to melancholic states at all periods of life; yet, the peculiar character of the psychosis is the frequency of religious despondency, and delusions respecting the moral well-being of the subject. The symptoms grow in severity; suicidal feelings become prevalent; and the delusion that the soul is lost often creates fits of mental agony or despair.*

At its early evolution painful mental states invariably prevail, and in over 55 per cent. of our cases mental depression existed throughout the attack—a subacute delusional melancholia being far the more frequent form; yet maniacal states are by no means unfrequent, and outbursts of excitement, alternating with depression, are prone to occur at a later stage of its history. Sensorial anomalies early arise and are strangely tinctured by the prevailing emotional gloom. The spirit-world is the subject-matter of her broodings; mystic communications are received from above announcing her hopeless fate, or threatening terrible judgments; or supernatural agents appear visibly and terrify her in her half-waking moments. Visual and aural hallucinations occur in about the same proportion of cases, 27 per cent. of the whole series being subject to such anomalies; whilst evidence of the implication of other special senses was very rarely obtained. Intellectual perversions soon ensue, sometimes evolved out of the sensorial disturbances, often independent of such states, but invariably intensified by hallucinatory and illusional phenomena when present. Delusional states were recognised in 73 per cent., and out of a total of sixty-one deluded cases, sixteen were victims to the terrible delusion that the soul was eternally lost, and that the subject was to be consigned to the flames of hell. It is strange to witness the prevalence of this religious despondency at a period when, as we are aware, the generative organs are undergoing an important cyclical transformation; and to contrast it with the converse states of religious exaltation so frequently associated with the sexual transformations and excitation of adolescence, of hysterical and epileptic forms of insanity. Usually the victim of this ailment accuses herself of the most heinous crimes, and dreads the pursuit of human or spiritual beings who thirst for her life's blood; but, at times, a case presents a somewhat different character of delusive belief; thus, one subject believed herself to be bitten by venomous toads, that dogs pursued her and sucked her blood; whilst another declared she was fed on human flesh. Such delirious concepts are by no means frequent in this form of insanity, nor are the patient's children or husband usually the theme of her perverted imagination.

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* Thus Dr. Skae characterised the alienation occurring at this period of life as:—"A monomania of fear, despondency, remorse, hopelessness, passing occasionally into dementia."

Such gloomy forebodings of coming evil or the mental disquiet aroused by a sense of her irrevocable doom, eventually issue in suicidal promptings, in fact, a large proportion freely admit this tendency. We find that of all the cases of mental depression taken together quite 60 per cent. are actually suicidal; but, of the class of patients now under consideration, about 44 per cent. only could be so considered. Impulses to suicide are certainly not so frequent; yet, in the worst cases, they do present themselves. Dr. Clouston expresses the opinion that:—"The very loss of courage and vigour of will operate against any effectual attempts at suicide"; yet experience teaches that in the worst forms most desperate attempts are occasionally made with the object of eluding the torture to which the mind is at times subjected by the terror of impending evil. Thus one poor victim of such terrors imagined herself haunted by evil spirits, who audibly told her she was to be burnt alive, and, consequently, she made a desperate attempt at hanging herself; another attempted strangulation under the impression of being pursued by the evil one; a third attempts poisoning with vermin-powder, upon the assumption that she was "cast off by God"; and three others attempt to end their misery by drowning, choking, or the knife, under the influence of similar perverted sensorial states. Especially have we learnt to dread the impulsiveness of depression at the climacteric when associated with the tendency to drink heavily.* The purely impulsive form of insanity may appear at this period of life; and homicidal, as well as suicidal, impulses may characterise the case, apart from any notable intellectual or emotional disturbance whatever. As we have before stated, the convulsive neuroses are prone to occur at all the cyclical epochs of life with special frequency.

S. H., aged forty-two, a married woman, in comfortable circumstances at home, was admitted suffering from severe mental depression. She was a strong, muscular subject of medium height, but somewhat pale and anæmic; had a family of three children living; and was last confined, some eighteen months ago, of a still-born child. Her habits of life were affirmed to have been consistent and temperate; and at the present time she was regarded as suffering from the functional ailments incident to the menstrual climacteric; the menopause was not established but much irregularity existed. Paternal ancestry neurotic; the father, who was regarded as insane, had committed suicide on hearing of his wife's death. For three months she had been melancholic, and had made repeated attempts to drown, choke, and suffocate herself, and also to cut her throat. Had expressed no delusion, and did not suffer from hallucination.

No cardiac or arterial disease was apparent; the respiratory and alimentary systems were normal. The urine was 1020 specific gravity, faintly acid, and devoid of albumen or sugar. Catamenia were in arrest. A few days after admission her morbid propensities declared themselves, and she attempted, by filling her mouth with paper, leaves, or anything at hand, to choke herself. Her restlessness at night was relieved by chloral, and the sedative solution of opium in 20 minim doses was administered twice daily. Her aspect was expressive of a sour discontent, and from being reserved, reticent, and brooding, she developed hypochondriacal fancies; and querulously and persistently drew attention to her imagined ailments. A slight attack of pleurisy a fortnight after admission was followed by marked relief to her mental symptoms, and she left perfectly recovered two months later. She had not been at home over a fortnight ere her restless depression recurred, and further suicidal tendencies led to her re-admission three months subsequently. Her condition was as follows:—"Free from any aspect of depression; utterly indifferent and callous to her existing state; admits, with some flippancy of manner, being subject to sudden and incontrollable impulses to destroy herself; no delusion or hallucination can be traced in her account of herself; she is perfectly calm, rational in all her statements, and intelligent. Amenorrhœa has existed for eight months." Under similar treatment she progressed during the first month so far as to be cheerful, active, industrious, and, according to her own statement, had suffered from no return of morbid impulse. She had, however, a somewhat careless, flippant manner, which was unsatisfactory as indicating a reduction in the moral sense not natural to her. About this time she again became somewhat querulous, importunate about returning home, and there was slight hypochondriasis. This culminated in her abstracting from a cupboard a quart bottle of spirits, locking herself in the bathroom of an officer's house, and deliberately swallowing the whole of the contents of the bottle. She was discovered in time to rescue her, and on her recovery from her semi-comatose state she avowed that she had taken the spirits with suicidal intent. Again she became more cheery, less hypochondriacal, active, and useful, and in six weeks time she was tried once more in an associated dormitory, where she slept with fifty other patients. She protested her freedom from impulses ever since her last attack, and was most anxious to be tried at home once again. The night following this apparently genuine statement, she retired to bed, and was found in the early morning strangled by a piece of braid which she had secured and concealed. The ligature was tightly secured, the bed-clothing perfectly undisturbed, and the sheet drawn over the face to secure her from the observation of the patients sleeping beside her—sufficient evidence of the desperate determination which had initiated the act.

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* See also Fleury, *Journ. Mental Science*, July, 1895, p. 551.

Contrasted with the case of *S. H.* we may take that of *A. A.*, in which maniacal perversions of a prominent nymphomaniacal nature prevail.

A. A., aged forty-seven, a married woman, of steady, temperate habits of life, without any ascertainable neurotic history in her antecedents, came under treatment after two weeks' excitement. She had only recently been discharged from another asylum. She was in a state of subacute excitement upon her admission, and was dangerously aggressive; she expressed the delusion that she was Queen, that her husband was king, and that she held the keys of heaven and hell; a suspicious tendency was also present which induced her to refuse her food. She was pale and somewhat haggard; a hæmic bruit was heard at the base; had a furtive suspicious expression, and an agitated eager manner; spoke of a contest about to occur in the country which she might be able to arrest. She admitted being at the menopause. For some weeks after admission the excitement abated sufficiently to allow her to occupy herself usefully. Soon, however, maniacal symptoms again supervened, and she has remained since this period subject to outbursts of excitement and intervals of calm. The patient entertained the most bitter feelings of animosity against her husband whom she repeatedly accused of being untrue to her; she denounced him in loud and threatening language, became most furious and violent, and in her frenzy destroyed all within her reach. Upon one occasion she tried to strangle herself by tying her hair round her neck. "She now betrays a strongly erotic condition evidenced by expression and gesture and lascivious remarks; when less under self-control this tendency gives vent to repulsive and obscene language. She has developed the delusion that one of the asylum officials is married to her, and she repudiates her former husband." It is now three years since the onset of her derangement; she is incoherent in conversation, betrays the same agitated manner, and is subject to periodic attacks of wild nymphomaniacal excitement as formerly.

Etiology.—In endeavouring to estimate statistically the influence of the climacteric involution upon the development of forms of mental alienation prevailing at this epoch, we naturally first question ourselves as to the relative frequency of insanity at this and other periods of life. Were we to take promiscuously an aggregate of cases becoming insane at different ages over an extended series of years, we should become early convinced that the question, far from being a simple one, as it might at first be regarded, was really a very complicated problem to unravel.

In the first place, the population at different ages of life varies; so that admissions in quinquennial or decennial periods are derived from disproportionate sections of the community. Hence we desire to learn not only the actual number of cases of insanity occurring at different ages, but also the ratio of such numbers to the population existing at similar periods of life. Thus a glance at Table on p. 429, showing the relative frequency of insanity at different ages for 1,808 female cases, admitted into West Riding Asylum, indicates the period from thirty to thirty-five as affording the highest number of admissions, and that each succeeding quinquennial affords a rapidly decreasing series of cases; yet, when we come to compare these numbers with the existing population at a corresponding age, we find, in lieu of a rapid decrease of insanity at these years, that the ratio of occurring cases is maintained at an equable rate or, according to some authorities, is even increased.

In the next place, our statistics would embrace subjects from most varied conditions of life and occupation, each sphere of life bringing its own disturbing and exciting elements into the field; hence it is advisable, in estimating the influence to be attached to the climacteric, to compare only that class of the community whose social status exposes them to more or less similar conditions of life. Unless this be done, we may drift into the error of attributing to a physiological cycle solely, what might more reasonably be expounded upon other grounds —the encroachment of environmental agencies. The wealthy and the pauper class (exposed as they are to such widely-different exciting agencies) must be judged apart upon the respective merits of their cases, for any such statistics embracing all sections of the community, irrespective of class distinctions, must be more or less vitiated thereby. In the next place, we must not forget that we are dealing with other important social factors—the single, the married, or the widowed state; and, that our aggregate certainly includes a number of relapsed and recurrent cases, in which a well-marked predisposition to insanity existed, and in which, therefore, the climacteric epoch simply supplies the disturbing forces.

In our attempts to classify such forms as appeared more especially dependent upon the climacteric change, we found it essential to eliminate a very large proportion of cases occurring at this period of life. Recurrent cases had obviously to be rejected, and so had all forms of insanity which had not originated at this epoch of life; and so also cases of epilepsy, imbecility, and organic brain affections, where the climacteric was but the spark to the fulminate. This rigid exclusion of dubious cases left but a meagre residue of eighty out of 1,808 cases or a percentage of 4·4 upon the total admissions. The

RELATIVE FREQUENCY OF INSANITY AT DIFFERENT AGES IN 2,277 CASES—MALES.

	BELOW 15.	15-20.	20-25.	25-30.	30-35.	35-40	40-45.	45-50.	50-55.	55-60.	60-65.	65-70.	70-75.	75-80.	80-85.	85-90.	90-95.	95.	Totals
Single, . . .	25	124	194	155	117	69	61	38	20	22	16	6	7	6	...	...	...	...	860
Married, . .	...	1	23	81	161	208	212	146	132	112	67	50	16	4	2	1	...	...	1216
Widowed, .	...	...	1	2	11	14	14	22	33	22	24	26	18	9	4	1	...	...	201
	25	125	218	238	289	291	287	206	185	156	107	82	41	19	6	2	...	...	2277
Census, 1883,	232449	59355	52398	48206	39971	36358	32620	26017	23052	15028	14681	10267	6286	3313	1301	312	39	7	...

RELATIVE FREQUENCY OF INSANITY AT DIFFERENT AGES IN 1,808 CASES—FEMALES.

	BELOW 15.	15-20.	20-25.	25-30.	30-35.	35-40.	40-45.	45-50.	50-55.	55-60.	60-65.	65-70.	70-75.	75-80.	80-85.	85-90.	90-95.	95.	Totals
Single, . . .	10	84	134	111	99	72	49	27	23	15	11	8	6	1	...	...	...	...	650
Married, . .	...	4	40	115	145	134	135	97	87	64	49	25	8	4	...	...	...	...	907
Widowed, .	...	...	...	6	11	17	26	26	39	42	32	22	18	10	2	...	...	...	251
	10	88	174	232	255	223	210	150	149	121	92	55	32	15	2	...	...	...	1808
Census, 1883,	225229	62992	56581	53523	43306	38763	35439	29053	25229	19725	16315	10742	6910	3786	1517	431	95	18	...

astonishing disparity in estimates upon this point by different writers is seen in the following table :—

Classed as Climacteric Insanity.

Reid's Hanwell Statistics of 703 Cases of Insanity.	Tilt's Statistics.	Skae's Edinburgh Royal Asylum, 558 Cases.	Clouston's Edinburgh Royal Asylum, 1,549 Cases.	Merson's West Riding Asylum, 1,054 Cases.	Bevan Lewis' West Riding Asylum, 1,808 Cases.
1·1 °/o	3 to 4 °/o	11·1 °/o	12·6 °/o	14 to 15 °/o	4·4 °/o

Obviously from the above the discrepancy must be involved in the *personal equation ;* in fact, in the want of unanimity of opinion as to what really constitutes the criterion of a so-called case of climacteric insanity. It will be observed that our estimate (founded upon personal observation) closely agrees with that of Dr. Tilt, and we differ, therefore, in regarding his results as an under-estimate ;* at the same time we glean from Dr. Merson's able paper † the real cause of such discrepancy. He there states in reference to his percentage of 15 that:—

> "It by no means follows that in all these cases the climacteric condition was the only, or even the chief, element in the causation of the mental disorder, though it may be affirmed that in most cases it exercised a causative or modifying influence more or less marked. The history of the cases investigated points to the conclusion that the change of life is not often of itself the immediate cause of insanity."

The same writer then proceeds to particularise 76 out of his 149 cases as influenced by other exciting agencies, giving amongst such some 31 cases of organic brain disease, cases of alcoholic excess, &c. Clearly such cases would not comprise pure cases of so-called climacteric insanity as understood by Dr. Skae, and with this qualification we fully agree with Dr. Merson's remarks on causation quoted above. Our own tables, which give but 4·4 per cent., include only such cases where the disturbances of the climacteric change and menopause were uncomplicated with other potent exciting agencies, and where we could justifiably presume that the insanity was the more immediate outcome of this revolutionary period.

In what way is this physical predisposition to insanity incurred? From the standpoint of the evolutionist, we are led to observe that the growth and development of the nervous-system is but a progressive representation and re-representation in ever-advancing and more complex terms of the whole organism ; that the cerebrum itself is but a vast assemblage of such highly-complex representative realms ; and that in the higher realms such nervous mechanisms as form the physical expression of such representation have linked to them the psychical correlatives of feeling and of thought. The loss of any portion of the organism which has entered largely into our conscious life, or, in other words, has been frequently, or at all largely, represented in consciousness, will necessarily disturb the mental balance. Nor could it be reasonably conceived that a portion of the body which had long subserved the wants of the organism, and whose physiological history was represented in certain organised tracts of the cerebrum, could suffer ablation without some attendant commotion in the brain. In fact, the systemic and least relational structures are in their genesis so closely interwoven with the physical substrata of feeling and emotion that wide-spread disturbance results from their derangements, functional and organic. The enormous share taken by the generative system in the physiological and psychological life of the female is a subject of paramount importance in our studies of the varieties of insanity. The organs subservient for some five-and-thirty years or longer to the important functions of menstruation, ovulation, gestation, lactation, find their nervous representatives in the fundamental tracts of the nervous system, and draw largely upon that system during their life of functional activity. Periodic relays of nerve-force demanded for the regulation of their blood-supply and of their muscular apparatus, and the ingoing currents crowding upwards from so extensive a system, enter intimately into the very web which forms the physical correlatives of emotions and moral instincts. The vast accession of new impressions registered by the sensorium when these organs awake to functional activity during puberty has a most profound effect on the mental constitution—an effect whose significance *cannot* be misinterpreted; for the result is a **real transformation,** more or less, of the **ego,** with all its feelings, emotions, sentiments, and desires. At each subsequent periodic crisis incident to menstruation, gestation, lactation, uterine involution, the nervous-centres are profoundly affected by the resultant transformations undergone, such periods being eminently periods of *nerve instability.*

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* *Psychological Medicine*, Drs. Bucknill and Tuke, 3rd edit., p. 360.

† "Climacteric Period in Relation to Insanity," *W.R.A. Reports*, vol. vi., 1876.

Pre-eminently is this the case at the menopause and grand climacteric, when the whole of this system loses its functional activity, degenerates, and, in fact, passes almost completely out of the life of the individual. Both subjective and objective accompaniments of the menstrual molimen in a greatly-exaggerated degree emphasise and usher-in this serious disturbance of the nervous-centres—headache, vertigo, faintness, "heat-flushes," emotional waves, phases of moral perversity, irritability, querulous impatience, even intellectual disturbance (especially of memory and of attention) prevail; and, wanting the relief afforded by the depurative process of menstruation, the distress is often a long-continued and urgent one. This, however, is the earlier stage of functional decrepitude, the early phase of which is characterised by want of decision, lassitude, and hebetude. It is essentially a period of voluminous emotions, purposeless waves of feeling, abortive yearnings, redundant, vague, uncontrolled desires, and misdirected energy.

That great reservoir of nerve-force, which had for its object the procreative functions of the organism, is now objectless, and its expenditure must now be directed into other channels; a period of emotional instability ushers-in a period of reconstructions.

J. F., aged forty-one, single; admitted January, 1880. Patient's father was very intemperate; her mother and brother had been inmates of this asylum. J.F.'s life had been a very unhappy one. For many years she had been compelled to dwell with her brother, a brutal, drunken scoundrel. During the two months before being taken to the asylum, she had developed dangerous, impulsive tendencies, suddenly striking, biting at, and kicking those happening to be around her. When admitted, a condition of fairly calm attention and apprehensiveness was constantly interrupted by outbursts of furious excitement and violence. An insane self-congratulation in her power of occasioning terror by her actions, seemed possibly of influence in the production of her paroxysms of passion.

For three months she was liable to these dangerous impulses, of which she was aware, and expressed her grief at being unable to restrain them. Subsequently she passed through phases of sullenness, depression, and agitation, to quietude and industry. The catamenia, absent on her admission, returned during her latter period of convalescence.

This epoch of reconstructions is one of peril to the mind, especially to those ill-trained mental constitutions in which the passions have been allowed an uncontrolled expression; and where intelligent guidance has been denied to the instinctive desires. In fresh objects of affection, in new pursuits, aims, and studies, in other forms of mental culture many minds will seek and obtain relief for these perturbed feelings and pent-up emotions. The anxious and intelligent mother will find a sufficient object in the prospective life and well-being of the offspring; many cultured minds will find in the fields of literature a sufficient relief to their pent-up energies; whilst those who lack in such facilities will perhaps bend their attention towards schemes of education or charitable movements; and the instances are not few where the political spirit of the times affords, in our day, missions for the same subjects. The peril of this period is one incident to all periods of reconstruction arising during emotional turmoil and perturbation. Reflection wants the calm essential to its orderly operation, and judgment is liable to be warped and one-sided; hence, also, it is that this age of life is one prone to bigotry, to religious fanaticism, or to conduct based upon dogmatic and immature beliefs. An unusual and inordinate religious zeal is, indeed, a most frequent expression of this transition-period in mental life; and this is of interest viewed in connection with the characteristic delusions of the insanity of this age.

We hear of a similar climacteric in man; but the parallel is more fanciful than strictly correct. Even its advocates who speak of climacteric insanity in man, allude to it as occurring "at a later time of life than in the female, . . . much more irregular and indefinite.

There is nothing to mark it off so clearly as the menopause" (*Clouston*).[*] The period assigned for the decline of the procreative power in man is 55 to 65; in fact, the borderland of senility and not a genuine epochal transformation. As Barnes states:—"there is nothing to compare with the almost sudden decay of the organs of reproduction which marks the middle age of women."[†] With a certain proportion of cases the menopause in women may, in like manner, usher-in premature senility; but, in all, its more or less sudden onset and the great constitutional changes and local transformations wrought, frequently followed by the subject taking up an entirely new lease of life, give to this period a critical character wholly distinct from what we see in man.

Prognosis.—The ultimate issue of an ordinary uncomplicated case of insanity at the climacteric may certainly be considered a favourable one; favourable, that is, as regards the duration of the malady, favourable as regards the stability of the reinstatement, and favourable as contrasted with the recoverability of *all cases* of insanity in the female when taken collectively. Even when all cases of insanity at this period of life are considered, whether of recent or of remote origin, complicated or otherwise, still one-half of such cases constitute absolute recoveries; and the favourable progress of the affection is indicated by the fact that three-fourths of the recoveries take place within nine months of the onset of the attack.

What, then, are the special features which serve to demarcate the favourable from the unfavourable class? what are the elements which enter into a favourable prognosis and the reverse? The hopes we can give to our patient's friends of a perfect recovery will largely depend upon the "*time element*," which plays so important a *rôle* in determining the prognosis in most forms of insanity. An early and rapid cure is, as in other cases, favoured by early treatment; and the chances of a complete and speedy recovery are much strengthened if the subject come under appropriate treatment within *two weeks* of the onset of her symptoms. We would emphasise here the *speedy* return to a normal mental vigour, for it is in this particular that early treatment is so desirable. The actual numerical result of recoveries coming under treatment, from a week up to three months after the onset of their symptoms, is about the same for all (50 to 60 per cent. being recoveries); but, beyond three months, fewer actual *recoveries* occur, and a large aggregate will be relegated to the class of the chronic insane.

Will the recovery be a stable one, or will the attack subject the patient to further liability? As a general rule, it may be affirmed that the more fully the affection realises the character which is regarded as typical of a truly epochal form of insanity—*i.e.*, the more fully it appears to be the issue of disturbances incident to this period of life—the more likely is the return to normal health to prove a secure and permanent state. Hereditary predisposition will, of course, in such cases produce its usual results, subjecting the victim at any period to a relapse on the incidence of exciting agencies; and the predisposing influence of former seizures will also have to be considered and allowed for in framing our reply to this query. An actual study of our cases of insanity at the climacteric shows that some 27·7 per cent. had a family predisposition to insanity; and that 38 per cent. of the total cases had suffered from a previous attack of mental derangement. Yet the actual relapse after confirmed recovery from this form of insanity occurred but in four instances out of the whole series of eighty-three, and in three of these a predisposition to insanity was indicated by a former attack in earlier life with strong hereditary taint.

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[*] *Op. cit.*, p. 560.

[†] *Clinical History of the Diseases of Women*, Barnes, p. 263.

Another point of importance in prognosis to recall is the mortality incident to this affection. The deaths, which amount to 14·4 per cent. at the West Riding Asylum, are due to intercurrent affections, in which phthisis or pneumonia play the chief part; in fact, one-half the deaths occurred amongst the chronic class who had resided at the asylum for a period of from two to six years. It may be stated, indeed, that the insanity incident to this period is rarely, if ever, fatal in itself; and the chronic remnant of this class owe their unfavourable character chiefly to the exhausting influence of chronic pulmonary disease, ulcerative affections of the bowels, or the malnutrition and defective blood-supply of the brain, due to an enfeebled and fatty heart. Another factor in the prognosis is the age of the patient. It has been observed by Dr. Clouston that fewer recoveries occur after fifty years of age—an opinion with which we concur.*

The previous habits of the patient must likewise be taken into account, and especially does this apply to the use of alcoholic stimulants, so frequently indulged in at this period of the woman's life. It is a well-known fact that secret drinking habits become peculiarly frequent at this era, a morbid craving for stimulation being engendered by the depression entailed at this period of reconstruction. If this vice has been contracted the malady always appears in an aggravated form. The very nature of the mental disturbance, the delusional melancholia often bordering upon a hypochondriasis, and always of a self-accusatory character, finds in this grievance a sure foundation for its gloomy fears and genuine despair; these cases, as already indicated, are peculiarly prone to suicidal impulse. Such subjects may exhibit much outward calm, have an absent manner, a self-engrossed aspect, or a suspicious furtive reticence, or betray on their features the set aspect of despair—indications which should place us on our guard.

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* *Loc. cit.*, p. 564.

The question of **time** during which the alienation has existed, the **age** of the patient, her **hereditary predisposition** to insanity, and the acquired predisposition through **alcoholic indulgence** are some of the chief factors which enable us to arrive at our prognosis in the case.

Very divergent views have been expressed with respect to the prognosis in climacteric insanity. Thus one authority says :—

> "Climacteric insanity is far from being a hopeful form of mental derangement."[*] Van der Kolk states—"If religious melancholy begins in the climacteric years, then the prognosis is unfavourable ;" whilst Dr. Merson says—"The history of the cases I have investigated, however, shows that as regards ultimate recovery the prognosis is by no means unfavourable, though an early recovery is not generally to be expected."[†]

Dr. Clouston gives a percentage of 57 for recoveries in the female sex ;[‡] Dr. Merson's table realising 59·5 per cent., or 47 per cent. when cases uncomplicated with epilepsy, general paralysis, and other brain diseases were excluded. Our later statistics, it will be seen, afford us a recovery-rate of 48 per cent., and, therefore, justify the views expressed by Dr. Merson.

What is the issue of the attack in the more unfavourable cases? It appears that about 36 per cent. form an incurable chronic residue, and about 14 per cent. meet with a fatal termination, half of which fatal cases also are derived from the chronic class. Yet these unfavourable cases do not necessarily demand asylum-supervision ; in fact, one-half at least become relegated to the home-circle again, and are able to discharge in a fairly-satisfactory manner the duties of the wife or mother, or compete for livelihood in their various spheres. Such incomplete recoveries are instances of a permanent mental enfeeblement, but are by no means subject to the recurrence of acute symptoms. They remain mental wrecks after the storm, the depth of reduction varying much for each individual case. Quiet, orderly, inoffensive, they need only the kindly guidance of the home-circle to keep them right ; yet, they exhibit an unwonted apathy, an indifference to former pursuits and pleasures, a lack of energy—mental and physical —which was present in their old selves. At times depressed, they never show active suicidal symptoms ; but exhibit a flabbiness of purpose and will, which render them for lifetime the dependents upon a stronger mind.

There is a remnant left, however, of these climacteric cases where the issue is far different, and where some of the worst forms of incurable delusional insanity become established. Here aural hallucinations largely prevail, and a sexual element often appears to enter into the material of their delusions. Hours are spent at the windows listening to the communications of these unseen agencies; to which passionate, wild outbursts of obscenity and abuse often succeed from the infuriated victim. Such attacks of excitement largely prevail at night; and the delusions based upon such sensorial anomalies lead to aggressive and destructive conduct. For many years these subjects remain a prey to their deluded fancies; are usually self-opinionated, or arrogant, overbearing, defiant in demeanour, and form a section of the more noisy and dangerous class in our asylum-wards. In such cases we can only hope for the speedy advent of a dementia before which the painful sensorial states and delusional perversions fade, while a settled calm and negative state of mind take the place of former turmoil.

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[*] *Psychological Medicine* (Drs. Bucknill and Tuke), p. 145.

[†] *West Riding Asylum Reports*, vol. vi., p. 107.

[‡] *Loc. cit.*, p. 563.

Treatment.—A tonic regimen is desirable in most cases of insanity at this epoch of life. Open-air exercise should be enjoined; a free nutritious diet devoid of stimulants; and careful attention to the *primæ viæ*. An aloetic purgative is often desirable at the onset, followed by mild laxatives, of which the mineral waters are a convenient form of administration. Our experience teaches us that a large proportion of cases recover without any form of medicinal treatment; the removal from their homes, the influence of new associations, and, above all, the strict attention to dietetic treatment sufficing to ensure a cure.

There are certain cases, however, where medicinal interference is imperatively demanded. Anæmia must be met by the administration of iron, preferably in the form of the *ammonio-citrate*, and in combination with small doses of the *liquor arsenicalis*. Iron should not be given if acute symptoms prevail either of the maniacal or melancholic stamp. We should await the subsidence of these symptoms, meanwhile trusting to agencies for ensuring sleep and to a liberal diet.

If there be a tendency to refuse food, we must not permit our patient to escape on the excuse of having partially taken her meal; a due amount of milk and eggs with beef-tea, nourishing soups, and farinaceous food should be rigidly insisted upon; and, if necessary to resort to force, compulsory feeding must be adopted.

Sleep must be secured by the administration of chloral, bromide of potassium or paraldehyd; of which we certainly give preference to the first. It is rarely necessary to give larger doses than 25 or 30 grains; and where from cardiac enfeeblement its use is inadmissible, paraldehyd may be substituted with good results.

The indiscriminate use of sedatives in these cases is, we think, to be deprecated; and, only in the more acutely-stamped types would we feel justified in the more continued use of sedatives. For this purpose chloral in combination with bromide of potassium is the safer treatment to adopt; opium or morphia, henbane and conium have proved unsatisfactory in our hands. The bromide given separately from the chloral we have less confidence in, and the combination found most desirable is 15 grains of chloral with 30 grains of bromide twice daily. We by no means share in the unfavourable view expressed by some as to the general inutility, or even hurtfulness, of sedative treatment in the acute forms of mental ailment at this epoch; given the means of securing efficient alimentation, such treatment is often followed by the best results.

SENILE INSANITY.

Contents.—Mental Derangements Incident to Senility—Senile Mania—Senile Melancholia—Chronic Cerebral Atrophy—Senile Convulsions—Senile Epilepsy—Senile Dementia—Inheritance as a Factor in Senile Insanities—Exhaustive Brain-work—Alcohol and Senility—Case of T. G.—Onset and Prodromata—Character of the Senile Reductions—Senile Hypochondriasis (J. A.)—Senile Atrophy and Thrombosis (I. B.)—Acute Senile Melancholia and Syncopal Attacks (H. D.)—Partial Exaltation in Senile Insanity—Delusional Perversions of the Monomaniac and Senile Subject Contrasted—Senile Amnesia—Case of Senile Insanity (M. M.)—Elimination of Urea in Chronic Cerebral Atrophy and Premature Senility—A Local Manifestation of Chronic Bright's Disease.

The student is apt to mis-apply the term *senile insanity*, that form of senile decrepitude which is but a morbid exaggeration of *physiological* senility; that gradual obnubilation of mind known as *senile dementia* is apt to be taken as a type of what is implied by senile insanity; and, so far is he right, that it is doubtless true a far larger proportion of cases of mental impairment in senility belong to this than to any other category of mental ailment. He is, however, too apt to assume that all varieties of mental ailments in the aged issue in senile dementia; and that the maniacal excitement which is so frequently observed at this time of life is necessarily the accompaniment or the precursor of senile decay. It is therefore, necessary to indicate that this term connotes a very large class of symptoms, embracing between them all the varied forms of insanity usually differentiated. The pathology of old age is as unique and interesting as that of infancy and adult life; and, just as we are aware, that certain periods of life bestow a special immunity from certain morbid affections, so do we find old age by no means an exception to this rule. That it has its own special affections of the central nervous system, as of the body at large, is also a well-established fact; and that such morbid changes are characterised by a special tendency towards *atrophy* has long been recognised.

Apart, however, from such special immunities and proclivities as this period of life is apt to entail, there are other forms of mental alienation common to it and to adult life which must be taken account of ere a faithful picture of senile insanity can be framed. Simple melancholic states, maniacal perversions, in themselves recoverable forms; or more obstinate delusional perversions with or without, permanent dementia, paralytic dementia, the dementia of chronic cerebral atrophy, epilepsy, and even general paralysis, may severally be encountered during advanced senility in the predisposed. Yet, it is none the less true that such affections, more common at other epochs of life, are considerably modified by the physiological stadium, and are stamped with a special impress which more or less distinguishes each form of senile alienation from the psychosis of earlier periods of life.

A careful study of insanity amongst the senile admissions into our asylums, and an attempt at a rational classification into groups according to their most obvious pathological indications, will cause the student much perplexity at first, owing to the extreme multiplicity of symptoms which he encounters amongst such cases. He will early learn, through the painful experience of a faulty diagnosis, that it is easy to confound functional derangements with the earlier indications of organic brain-disease; and he must be fully prepared to find his prognosis stultified, unless due attention be paid to the modifying influences of the senile epoch over the nature and course of the disease.

Senile Mania.—He will meet with forms of simple maniacal excitement without any very obvious enfeeblement of the intellectual faculties, in which emotional instability, incessant garrulity, and restlessness are the only obvious disturbances recognised. Such excitement may vary from one of slight degree to very acute forms; and, in the latter case, may prove most persistent and most obstinate to all remedial agencies. The rambling disconnected speech may pass into utter incoherence, the motor restlessness become extreme, and insomnia defy all our means of relief. Such cases of senile mania may require long-continued and forcible feeding, and cause us much anxiety lest a fatal degree of exhaustion ensue. And yet, such cases, although often the precursors of permanent dementia, may completely recover and leave our patients with scarcely a vestige of mental enfeeblement apparent, beyond what is natural to their time of life. It is to the distinction between such recoverable forms, and those intercurrent attacks of mania which are frequent during progressive senile atrophy of the brain, that the student will have his attention chiefly directed. Recurrent maniacal attacks are of special frequency amongst the aged insane, who are prone to explosive discharges from their ill-nourished and highly-unstable cerebrum; and such recurrent seizures are notably present in those senile cases who have acquired a predisposition through alcoholic indulgence.

Senile Melancholia.—A second group of cases is presented in those forms of simple melancholic depression to which certain predisposed subjects are liable during the physiological involution of the nerve-centres on the advent of senility. Depression at this epoch is always of most ominous import; it may be the precursor of senile mania, it may usher in hopeless forms of senile dementia, or it may be the warning-note of those serious forms of dementia which are connected with an interstitial or gross cerebral change, such as occur in chronic cerebral atrophy with its scleroses, or hæmorrhages, or softening from thrombi. Here, again, the student will find his attention profitably directed towards the diagnostic distinctions betwixt simple senile melancholia as a purely functional ailment (which is a fairly recoverable form of alienation), and the depression which augurs a serious structural change in the nerve-centres. As we shall see later on, such forms of simple melancholia, unaccompanied by any delusional state, are notably characterised by their strongly-marked suicidal tendency, which appears in 79 per cent. of such cases.

Chronic Cerebral Atrophy.—Passing by these simple forms of affective insanity, we arrive at a third, and a very interesting, group of cases, which, whether the symptoms be considered from the mental or physical side, present evidence of a very definite pathological process—we allude to chronic cerebral atrophy. This is an affection no more limited to senility than the foregoing, yet it is of special occurrence at advanced age. The affective sphere of mind is also here involved, and painful mental states predominate. Despondency and gloom issue at times in acute melancholic or maniacal outbursts, yet morbid depression is here usually associated with enfeebled will and a special tendency to instinctive, impulsive states. Chronic melancholia, with impulsive propensities, is the prominent mental symptom. The disease appears to project itself mainly upon the motorial sphere of mind; and the diseased vascular tracts in the cerebral hemispheres are peculiarly prone to appear in the frontal and motor realms, cortical and ganglionic. A group of symptoms constituted by despondency, self-absorption, general intellectual torpor, failure of memory, enfeebled volition, impulsive conduct, often desperately-suicidal attempts; and, with this, highly-characteristic physiognomical signs, such as a pained, vacant aspect, a dulled, lustreless eye, a jaundiced earthy tinge of integument, tortuous radials, temporals, or brachials, a slow and laboured utterance, and an utter want of initiative—are in themselves a very suggestive category. When, with such a case, we find general muscular enfeeblement progressively advancing, a history of slight, transient strokes, a very temporary loss of speech, a slight glossoplegia, facial or brachial monoplegia, or slight syncopal or vertiginous attacks repeating themselves frequently, we may be pretty confident that we have to deal with a case of chronic cerebral atrophy as the result of vascular disease, and probably associated with considerable renal and cardiac degeneration.

Senile Convulsions.—A fourth group presents itself under the form of convulsive affections. After a more or less prolonged period of mania or melancholia, the persistence of which may be unexplained, there will suddenly occur a partial or general convulsive seizure, with, or without, loss of consciousness. The patient henceforth becomes subject to more or less periodic attacks of genuine epilepsy or epileptiform convulsions. Such cases, however, are not so numerous. Out of 260 cases of senile female admissions but 20 cases, or 7·6 per cent. of the whole, were subject to convulsive attacks, and in not more than one-half this proportion did the convulsive neurosis assume such a grave aspect as to be regarded as the most prominent morbid feature.

Senile Epilepsy occurring during the course of any form of mental alienation is, of course, of very evil augury. It betokens, usually, a localised nutritive derangement of a grave character, due to diseased vascular tracts of the cortex or ganglia at the base.

Senile Dementia.—Lastly, there is the well-known insane dotage of the senile dement, in which all the mental faculties are progressively affected, and in which maniacal excitement, or, less frequently, melancholic gloom or agitation, may recur over and over again, and very vivid hallucinatory and delusional conditions may prevail; yet a steady decadence of mind proceeds and utter fatuity results.

With respect to the question of inheritance in senile subjects, let us remember that although much depends on the organised stability of the nervous-centres, we must not omit to lay due stress upon exceptional environmental conditions of life, and especially upon conditions self-induced, or to which the organism wilfully exposes itself in defiance of all physiological dictates. Whatever be the resistance of the organism to morbid excitants, we can safely assume that the latter *may* reach such an intensity as to break through all opposition, and that inherited instability need by no means be predicted in such cases; in other words, insanity may be *acquired* purely *ab extra.* We have too many instances afforded us of the break-down of good, stable minds through the stress and tension induced by surrounding conditions of life, to permit us for one moment to hesitate in our acceptance of, or to allow us to qualify, this statement. Sustained mental exertion may be carried on under unfavourable circumstances to such a pitch as to issue in complete demoralisation; undue stimulation of nervous-centres already fagged by overwork, will as certainly, if persisted in, entail in the issue complete disorganisation, or deterioration of the output; it is, therefore, highly necessary that we should lay full emphasis upon the environmental conditions. Given a case of strong hereditary predisposition, and we infer that slight exciting agencies will suffice for a culmination in some morbid development. Given but a feeble predisposition, and the resistance to morbid excitation rises; so that a *potent* cause only *ab extra* will induce the vicious evolution. Yet it is equally true that, apart from any traceable ancestral frailty, certain vicious conditions of life will of themselves induce such cerebral disorder as to culminate in an attack of insanity. What do we know respecting ancestral history and the interaction of the environment in cases of senile insanity?

A predisposition was clearly ascertained in some 58 cases of senile insanity out of 261 male patients, or a percentage of 22. This estimate includes collateral and direct transmissions, and was limited almost exclusively to the parentage and to the collateral line of brothers and sisters. If *direct* inheritance only be taken into account, the percentage would still remain as high as 15. Now this is considerably above Dr. Clouston's figure (13 per cent.), and, in fact, lies midway betwixt the percentage given by him* and the average heredity ascertained for 1810 cases of all forms of insanity alike (31·5 per cent.). Dr. Clouston speaks of ancestral inheritance as "*very uncommon*," but admits that such estimate includes a fallacy "that the facts about heredity were further back and more forgotten in this than in any other form."† Such a qualification, undoubtedly well-based, applies equally to our own statistics; and we may with justice assume that an insane inheritance would, if all the facts were forthcoming, be found to be pretty much what is the average for all forms of insanity taken together.

But apart from the *frequency* of its occurrence as a factor is the question of the nature of such inheritance and its intensity; unfortunately our data for a reliable conclusion upon these points are too scanty. Dr. Clouston's assumption is that—"To have survived, therefore, the changes and chances, the crises and perils of life with intact mental function till after sixty, means slight neurotic heredity, or great absence of exciting causes of disease."‡ Were we to regard the dictum that the stronger predisposition is manifested earlier in life (open as it is to such numerous exceptions) we would still take exception to applying such a law to the cases of insanity under consideration. What are the positive facts before us? If we take into consideration all forms of neurotic inheritance alike, we find a percentage of 26·4 give such histories; that in several cases both father and mother were insane; that in some, several members of the family were epileptic; that in others, direct hereditary insanity was traceable (associated with epilepsy and paralysis), and that suicide was not infrequent in the family. We should, therefore, incline to the view that the senile insane exhibit a fairly average predisposition to insanity; and that, possibly, its late development in such subjects may depend upon the *nature* of the neurotic inheritance and the developmental period during which it was originally acquired by the ancestor; for the law that a morbid condition tends to reappear at

* *Op. cit.*, p. 567. † *Ibid.*, p. 566. ‡ *Ibid.*, p. 566.

an earlier age in the progeny is not final upon this point. For us, however, the more important point for recognition is that whatever proclivity towards insanity there be in such subjects, due to inheritance, there is a most powerful agency in operation in a large number of such cases in the surrounding conditions of life. Undue cerebral excitation, whether in the form of excessive mental work, and especially when prolonged intellectual operations are associated with anxiety and worry, or exhaustive emotional states, frequent exhaustive demands upon the intellectual operations, sustained mental tension in the struggle and competition for existence, will, as we well know, result in utter mental and physical prostration, even in those who possess the elasticity and resistance of manhood; much more should we expect such agencies to be operative for ill, at an age when the brain-cells have reached their limit of normal functional activity, when function declines, and physiological dissolution commences, in the series of downward retrogressive changes of senility.

Such unwise demands made on the nervous-centres during adult life are prone to induce premature senility, and the various mental derangements to which the aged are subject; but a still more potent factor is comprised in the association with these conditions of alcoholic stimulation. Excessive alcoholic indulgence lends a frightful impetus to the retrograde changes of this epoch, tending to over-excitation and exhaustion of the nerve-cells; to retention of hydro-carbon in the system; to vascular paresis and disease of the arterial tunics; to universal degradation in type of tissue, notably of the nerve-centres: we may, indeed, readily appreciate the evil effects of undue mental exertion backed up by alcoholic stimulation.

T. G., aged fifty-two, married, with a family of five children, a miner by occupation. Patient was suffering from his first attack of insanity, which began a month ago. He was a short, well-nourished subject, of florid complexion, conjunctivæ tinged; arcus senilus well marked; pupils equal and active; slight flattening of right side of face; tongue protruded straight, slightly tremulous. There was excessive sensibility of the soles of the feet; when these were tickled, the whole body was thrown into a convulsive state. He had formerly been addicted to very heavy drinking, although more abstemious during the past four years. His father drank to excess, and was a "bad character." His father's sister cut her throat at this asylum. Patient's two sisters (twins) are at present inmates here. Mother was at this time seventy-five years of age, strong and hale. No history of cranial injury.

About eleven months ago, patient had noticed convulsive movements of both eyeballs (*nystagmus*), and, subsequently, jerking movements of the head and neck. He sought treatment for his eyes at the Leeds Infirmary; but, whilst there, his limbs suddenly one night were thrown into convulsive movements like the head. Such movements recur frequently, but have sometimes left him completely for a week together; they are always increased during emotional states. He had *gradually* lost power in his right arm and leg; and speech also had become much impaired. Childish moods were then noticed; he would collect worthless rubbish, and, if deprived of it, become most passionate and violent. Memory deteriorated, and he began to misname his acquaintances and relatives. Great irritability was apparent later on; he would violently assault little children without obvious reason.

A fortnight before admission he had, for the first time, a succession of fits, in which "his eyes were drawn-up—he frothed much at the mouth, his mouth being drawn to one side (which side unknown); his limbs were much jerked about, and he remained for some time unconscious." Had suffered lately from aural and visual hallucinations; accused his wife of being unfaithful to him, and said, "people make a fool of me." He had threatened his wife with violence, and had said he would cut his throat.

He was emotional upon his admission, singing, weeping, and rambling alternately. He was then obviously suffering from ataxic aphasia, with a certain degree of amnesia. "Asked to pronounce a certain word, he frequently repeats the first syllable before proceeding, and when the word is completed successfully, he is apt to reiterate the whole word over and over again, or interpolate it subsequently into a sentence wholly irrelevant and foreign to it in context."

"He understands all that is said, but his replies are frequently quite incomprehensible; memory is notably impaired, and consciousness so far affected that he fails to recognise where he is, when he came, or the nature of his surroundings. He shows the *instantaneous obliteration of memory* for any given name or date so characteristic of certain alcoholics.* He still evinces distrust of his wife, and is inclined to impute to her all his troubles and present physical ailments. Attention is commanded with fair ease. He becomes readily emotional and agitated, when his breathing is accelerated, and choreic movements of head and limbs supervene; locomotion somewhat impaired by the jerky movements of his limbs."

Such being the history prior to and upon admission, the patient, a fortnight later, had several recurrences of the choreic movements, became decidedly suicidal, dashing himself head foremost on the floor. Three months later, he had a paralytic seizure sudden in onset. He fell on his knees; was unconscious for a short time; the right side of face and right arm showed muscular twitchings—the eyes turned to the right side, and the eyelids were convulsively affected. The twitching of the right arm continued long after return of consciousness; slight right hemiplegia was present the following day.

For several weeks he continued very restless, in a heavy, stupid state, and evidently much demented; both pupils minutely contracted. The limbs became more enfeebled, and he had to be kept in bed.

A recurrence of the convulsive attacks supervened some months later, and after lying for a few days in a semi-conscious state with constant twitchings of the limbs, and a high temperature, he died.

Sectio cadav. Skull-cap symmetrical, of average thickness and density; no adhesion of dura mater. Sinuses contained fluid blood. Arachnoid opaque over frontal and parietal regions, with considerable fluid in the meshes of pia mater, which was *thick* and *tough*, but *stripped freely*. There were one or two doubtful patches of adhesion along the marginal or first frontal gyrus. Vessels at base were *very atheromatous*. There was great wasting of gyri in the frontal and parietal regions, *especially on the left side*. The brain throughout was softer than normal, and of a dirty, rusty hue. The grey matter was shallow; the white matter studded with very numerous coarse vessels, showing also upon section small patches of a *pinkish tint*. No special focus of softening was noted. Cerebellum and basal ganglia presented no change.

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* See case of *J. F.*, p. 348.

Whole brain weighed	1270 grms.	Left frontal lobe weighed	198 grms.
Right hemisphere ,,	555 ,,	Cerebellum ,,	167 ,,
Left ,, ,,	515 ,,	Pons ,,	20 ,,
Right frontal lobe ,,	327 ,,	Medulla ,,	8 ,,

The heart weighed 355 grms.; muscle pale, ill-nourished; large patches of atheroma at base of aorta; valves healthy.

Right lung, 955 grms.; intensely engorged throughout, but still floated, and on pressure crepitated; no tubercle or inflammatory induration.

Left lung, 810 grms.; adherent by old fibrous bands; like the right lung, it was engorged throughout.

Liver, firmly bound to diaphragm; substance firm and fibrous.

Spleen, 163 grms.; congested and friable.

Right kidney, 140 grms.; left kidney, 180 grms. Capsules were adherent in both organs; the surface granular; the substance greatly wasted in cortical and pyramidal portions; the pelvis of the right kidney was dilated.

Of the 261 male senile cases, as many as 75 (*i.e.*, 28·7 per cent.) were conclusively proved to have been of intemperate habits for some years prior to their attack of insanity; and there is reason for regarding this percentage as far below the actual truth. Later on, we shall find that alcohol gives a special direction to the morbid tendency, having a preponderating influence in the production of special forms of senile psychoses. Thus senile mania, melancholia, and the dementia of chronic cerebral atrophy each afford an alcoholic history in 40 per cent.; whilst amongst the senile dements proper, this factor appears only in 16 per cent.

Onset and Prodromata.—Were we to attempt to define the boundary betwixt the physiological and pathological form of senility, between the ordinary second childishness of old age, and the dementia resulting from the senile atrophy of disease, we should find the task a difficult if not an impossible one. No such limit exists; the one form passes by such gradations into the other, that it is, at times, impossible to say that the physiological retrogression has been respected, and that the symptoms imply no genuine pathological change. Cases there are where the onset of senile dementia is so marked, or so sudden, or so premature, that no doubt whatever can be entertained that the physiological barrier has been overstepped; yet, in most instances, the atrophy of premature senility, at its onset, heralds itself by very uncertain symptoms, which pass by insensible gradations into the less equivocal character of the fully-developed disease.

Amongst the prodromal signs of this affection are an uncertainty and fickleness of disposition, and rapid changes of mood. Moody *taciturnity* alternates with fits of almost childish hilarity. The patient exhibits unreasonable irritability, spasmodic passion upon trivial occurrences, intolerance of contradiction or restriction, and impatience of former pursuits. Dr. Anstie has drawn attention to the irritable perversity of early stages.* Hebetude and lassitude are frequent precursors of incipient cerebral atrophy; but, the more striking feature is the alternation of moods, emotional variability and explosiveness. Headache is often a prominent symptom, and vascular turgescence a notable feature; slight exertion, such as ascending a hill or a flight of stairs, or violent laughter, resulting in swollen contorted veins and florid face. Then arise noticeable defects in the intellectual operations due to occasional lapse of memory, very occasional and very transient, yet anxiously noted by watchful friends. Insomnia at night may alternate with diurnal hebetude, lessened activity and languor.

Symptoms.—Such signs are of ominous portent in those advanced in years, or whose former mode of life is known to have been favourable to premature senility. An attack of maniacal excitement may now usher-in unequivocal signs of the nutritional impairment of the brain; and upon its subsidence, well-marked indications of enfeebled mind appear.

Similarly, much mental depression may ensue, and melancholic agitation precede the more profound reductions of a later date. In the greater number of cases, however, the transition is a gradual one, from the prodromal signs indicative of failing nutrition, to those of functional derangement of greater gravity, or of its complete arrest.

The failing mental powers illustrate the law of dissolution, whereby the highest and last-evolved members of a series fail earliest. The power of *abstract thought* suffers early; complex reasoning becomes a painful effort; mental processes generally become simple and more automatic. Representative states are less vigorous, and association of ideas enfeebled; hence, the contrasting faculties of the mind lose their former energy. The creative operations of the imaginative sphere decline, and reverie usurps their place in the mental life. The higher emotional states and moral sentiments fail to affect the mental life and the conduct of the individual with the vigour of former days; and, in fact, the possible adaptations of the organism are far less complicated and its environmental horizon is correspondingly limited. This may all be true, and yet memory may not be greatly affected; sooner or later, however, this faculty declines, and, as has been frequently observed, the failure is chiefly as respects *recent events*, the *more remote* events of the history being recalled vividly and accurately. We do not here recognise the instantaneous loss of impressions referred to in alcoholic cases; it is not a feature in senile insanity apart from alcoholism; yet, that there is greatly diminished impressibility in senile dementia is nevertheless true. The characteristic senile memory, the diminished revivability of recent impressions as compared with more organised ones, betrays itself in the whole tenor of the patient's life henceforth; he lives his childhood's days over again; recent impressions have but a transient and faint influence upon his ideation—or they fuse with the more vivid series of older and more remote states of feeling; a dreamy reverie takes the place of vigorous perceptive processes; byegone events appear transformed into existent realities, and are blended and confused with the passing events of the moment. Localisation in time and space will eventually be impossible, and complete incoherence of thought will ensue. Although this law of dissolution is invariably exemplified in senile dementia, it is remarkable that at times we are surprised to find, even in cases of profound enfeeblement of memory, a transient gleam of intelligence—the recognition of a series of perfectly recent impressions in their natural connection—when we had supposed the subject was completely oblivious to such circumstances. This seems only explicable on the assumption that the law of *trivial association* will bring together the recent impressions of the moment in relation to the more deeply-organised states of the past; and that the more deeply-organised are the states with which such recent impressions are associated, the more recoverable such states of consciousness are and the more resistant to morbid influences.

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* See *Psychological Medicine*, Bucknill and Tuke, p. 342.

Now follows a greater or less blunting of the special senses; deafness is a common symptom; there is also slowed nervous conduction and a sluggish reaction. The **expressive** faculties suffer likewise with the **impressive** sphere; not only is the subject less recipient and impressionable, but he is also less reactive. The intelligent initiative is rarely assumed; all actions are more instinctive, automatic, and impulsive; speech is hesitating, slowed, and highly characteristic—not only as the result of dementia; but also because the tongue has lost its cunning and is less glib; there is a dislike for any mental effort from the outset, and eventually an utter inability for sustained concentration of the mind on any one subject; there is an equal distaste for *physical* exertion. Reduced to an automaton, in every sense of the word, his habits of life are simple in the extreme; and all his feelings, thoughts and utterances savour of sameness and repetition. Of all the insane, the senile dement is the one whose **habits of life are most stereotyped,** and whose actions generally and speech are least variable and, therefore, most predicable.

In the dissolution of his nervous organisation the doubly-compounded rhythmic actions of an elaborate nervous mechanism appear to have given place to the simpler rhythms regulating the activities of simpler forms of life. The physical signs of senile decrepitude are notably marked; they are those of almost universal atrophy. Exceptional cases occur where obesity prevails; but the rule is that all the tissues, viscera (except heart and kidney), and glands undergo excessive atrophy, often preceded by fatty degenerative change. It is scarcely necessary to more than recall the thin harsh skin; the wrinkled face; edentulous jaws; the senile *arcus;* the grey hair and bald pate with its glossy atrophic integument; the tortuous corded temporals or radials; the diminished stature and weight; the skinny, shrunken extremities; evident emaciation, muscular enfeeblement, and wasting; stooping attitude and tottering gait.

It must not be supposed that the mental decadence here sketched-out takes place without emotional storms; for wild gusts of excitement sweep over the scene repeatedly during the progress of the cerebral atrophy. Such maniacal attacks are often most persistent and most obstinate to treatment. In fact, the recurrent maniacal attacks of senile insanity during the progress of cerebral atrophy are the *least amenable to treatment of all forms of mania.* The long continuance of such excitement, the deprivation of sleep, and incessant restlessness, is often a source of surprise to the student of insanity, who would *a priori* anticipate rapid exhaustion as the natural outcome. Such cases naturally cause much anxiety and trouble to their guardians, but this anxiety is largely amplified by their occasional refusal of food, and obstinate resistance to compulsory feeding. The maniacal outbursts of these senile dements is often revolting from their degraded habits and utter disregard of all decency. They are filthy in the extreme; they are also destructive, and are continually removing their clothes and exposing themselves. A sexual element, also, is often a prominent feature in such subjects; erotic tendencies being by no means infrequent during such maniacal attacks. Thus it is that at the onset of senile insanity, ere other well-pronounced symptoms declare themselves, the subject (formerly a most moral and well-conducted man) suddenly exhibits such erotic tendencies, outrages every sense of decency, and brings disgrace upon himself and connections, ere it is discovered that he is an irresponsible agent.

This objectionable tendency is one strong reason why an aged parent, suffering from such maniacal outbursts, should be removed from the care of his family. Occasionally such outbursts of excitement alternate with depression, and the following instance of senile hypochondriasis illustrates this well:—

J. A., aged sixty-two, widower, a labourer; a man of average height, and well-nourished, with a bullet-shaped head, light sandy-coloured hair, and blue eyes. Patient had lived a steady, temperate life; had suffered from no special illnesses—"stroke" or "fit." Had sustained no injury to the head. Family history was not ascertainable. He had been wretchedly depressed for six months; but for three years preceding his admission, he had suffered from slight attacks of depression alternating with excitement, and was taken charge of in the workhouse infirmary-wards. Six months since, whilst in a very dejected state of mind, he sprang over a bridge into the river, but was rescued and taken to the workhouse, where he has remained employing himself at useful occupations, but *always depressed*. After an exciting religious service which he attended six weeks prior to his arrival at the asylum, he became terribly dejected, and, at times, acutely melancholic, rushing about, wringing his hands, and crying out, "lost, lost," and tried eventually to leap from a window. He was brought to the asylum in a state of great agitation, struggling wildly, and necessitating restraint during the transit. The excitement abated shortly afterwards; he took a hearty meal and slept well. The next morning he was able to converse calmly, and gave a detailed account of his past life. "For some years past he has been subject to periods of 'confusion of thought,' and failure of memory; it comes on *suddenly, unexpectedly*, and at such times he is wholly incapacitated for work. At times such attacks were prolonged and accompanied by intense depression, during which he had to be placed under supervision. In one of these fits of despondency he attempted suicide. He believes he is 'eternally lost;' but his chief trouble at this time was not so much his soul's welfare, as the idea that he cannot digest his food; he constantly dwells upon the condition of his stomach and bowels, to which he refers many imaginary ailments." His memory was but slightly impaired, being more sluggish of recall than actually defective; his intellectual operations generally were torpid; his attention enfeebled, so that occasional confusion of ideas occurred during conversation; no hallucinations existed. He had a dejected expression. The pupils were equal and active, of normal size; the tongue was protruded straight, and showed no ataxy or tremor. No evident degeneration of the superficial arteries was indicated, and the heart was apparently healthy.

Chloral alone afforded him relief, and was frequently required to secure sleep. Thus he remained for some years with no relief nor alteration of his deluded state, pacing up and down the wards with woe-begone aspect, groaning, importunate upon the subject of his hypochondriacal fancies; dwelling upon his "depressed spirits," "sleeplessness," and "the fire that has for twenty years burned within him." He died, seven years after admission, of pulmonary congestion.

Many cases of senile dementia present for some time a gradual, progressive mental enfeeblement, without any notable degree of excitement. A constant unrest, a mild depression, with very vague fears and ill-defined notions of coming trouble prevail, often associated with distrust and more definite suspicions of persecution. Such was the following case, in which the ideas of persecution became more defined, and issued in paroxysms of great agitation; and in which the progress of atheromatous, vascular disease resulted in a slight transitory monoplegia from plugging of the vessels.

I. B., a widow, aged seventy-five. Lost her husband eight years ago, and lived with her children until removed to the workhouse, nine weeks since, having become unmanageable at home. She had been failing in mental powers for some few years. At this time she was excited and very restless, wandering about her room day and night, seeking her husband, whom she believed still lived, and expressing fears that the inmates of the workhouse had designs upon her life. Her habits as regards alcohol had been temperate; there was no history of insanity or other neurosis in her antecedents. Upon admission she was fairly quiet, and conversed affably. Grave mental failure was apparent; she still thought she was at the workhouse, although she had been here a week; "came direct from her own home, where she had been living with her husband and mother-in-law; the latter is not old, and she speaks of her husband as youthful; she herself is about thirty-six years of age; her husband lives in New Leeds; she saw him a fortnight ago." "She makes vague statements of ill-treatment before coming here, weeps childishly, and then as suddenly becomes cheerful and contented. She is a shrunken old woman, much reduced in bodily condition; the face much wrinkled, the complexion earthy, and the hair grey and scanty; there are no teeth. The superficial arteries show no marked signs of degeneration; the pulse is 90, regular and of fair strength; no obvious morbid state of circulatory or respiratory systems appreciable; urine almost colourless; specific gravity, 1010; no trace of albumen; no deposits." During her first month's residence here she exhibited the usual reductions of the senile dement, but no outbursts of excitement occurred. She slept soundly and took her food heartily, yet she was restless, discontented, peevish, wandered about in an aimless, half-vacant manner, and, when questioned, explained that her husband was at work close by at a colliery, and that she sought him and her son. Her habits also were inattentive; did not recognise the nature of her surroundings; believed she was still at Clayton Workhouse. About four weeks after admission she had one of the characteristic seizures to which these aged dements are subject. Whilst sitting in her chair she became pale and faint, lost consciousness momentarily, and the left upper extremity was found paralysed. The patient remained in a state of semi-stupor for about an hour, then recovered rapidly, and shortly afterwards could walk about readily; the paralysed limb regained its usual power. A month later it is noted that "she fancies her father lives here, as also that his age and her own are the same. Is dirty, restless; often weeps and cries to go home; is more feeble and wasted." Still later on she became melancholic, even acutely so; appeared in great agitation and terrified; would cry aloud, "Are you going to put me underground?"

The following case is one of a more acute character; the prevailing mental state being that of melancholic depression, attaining at times a degree of acute melancholia. It will be noted how the physical examination testifies to the general malnutrition, great emaciation, and to the failure in cardiac energy and advance of atheromatous change in the peripheral blood-vessels. The most striking feature in this case is the hypochondriacal state, which is not at all unusual in the acute forms of senile insanity; hideous delusions being entertained as to the sinister intention of those with whom the patient is brought in contact. The rapid cardiac failure often leads to alarming syncopal attacks.

H. D., aged sixty-two, and married. A somewhat tall woman of very haggard, wasted aspect; the complexion sallow, and condition generally very anæmic. Physical examination betrayed enfeebled cardiac energy; the impulse exceedingly weak, the sounds at base less clear and sharp than normal, but there was no murmur. The temporal and radial vessels were somewhat hard and incompressible, but not tortuous; pulse 96, very small and feeble. The abdomen was shrunken and hepatic dulness much diminished. Urine 1020, slightly cloudy, amber-coloured, alkaline; a trace of albumen; a large deposit of mucus. The patient on admission, was very restless and maniacal—suddenly springing out of bed, knocking over the buckets of water used by the scrubbers, and upsetting the patients' food, declaring it to be poisoned. She made strenuous efforts to escape from her ward, refused her food, asserting that her body was "full up," and there was no room in her stomach for food. After a restless night she was found very melancholic, and the subject of hypochondriacal delusions. Thus, she stated that for two years past debility and loss of appetite had prevailed, and she had now discovered that her spine had grown forward so as to encroach upon her stomach, preventing the entrance of food; her bones had grown longer and thinner, owing to a poison in the water in which she bathed herself. She affirms that stars appear in the room, talks childishly, and falls into a doze. Made such remarks as—"Don't put my eyes out; are you going to blind me? are you going to make me deaf?" Occasionally her melancholic fears so got the better of her at night that she could not be kept in association with others, but had to be removed to a single room, and a small dose of chloral administered; cried out in terrified tones—"Don't do it to-night, will you? What *are* you going to do? you have done it long since, haven't you?" Her habits were very degraded. A month after admission she had a *severe syncopal attack*, remained very pale and subconscious for some time, and the pulse became extremely feeble. She died of pneumonia shortly after this date.

When mental exaltation prevails it is a symptom of gravest moment and ominous of coming dissolution; the general exaltation, which is the prevalent feature in all simple maniacal states, is but the expression of a very universal disorder of function. It is the partial exaltation manifested as regards one or other of the most complex groups of feelings which comprise the æsthetic or the religious sentiments, the social and the domestic feelings, the sentiments of possession, of freedom, and many others—the later developments of civilised man—it is this exaltation which is so ominous of coming evil. The sentiments are, as expressed by H. Spencer, purely re-representative feelings; a compounded aggregate of numerous recollections, vague yet massive, combined with a still vaguer part, a kindred feeling organically associated by ancestral experience.* Such optimism is usually the knell of commencing decay. We need not here attempt to analyse such complex groups; we need only recall the fact that according to the special group we are dealing with, there will be more or less of the emotional or of the intellectual constituent; that the higher the developmental phase such feelings have attained, the more closely will the emotional and intellectual factors be interblended, and, therefore, the less distinguishable *inter se*; and the more preponderating will be the cognitive elements. The cognitive element is the *relational*; for thought is but the passage from one state of consciousness to another; it is, in itself, but a *feeling*, which is of insignificant duration, as compared with that of the states of consciousness which it connects. In the *sudden* transition from one state to another, we get a more pronounced relationship; the cognitive element becomes *more emphasised*, and thought more definite. Hence, the import of the fact that the relational element of mind is the first to succumb to mental disease. If, as may well be conceded, the change in the nerve-cell represents the physical basis of conscious states, and its protoplasmic extensions represent the relational nexus between cell and cell, whereby a change from one state to another is rendered possible, then our statement is tantamount to affirming that there is a failure in the forcing of the relational channels—that a greater conscious effort is required for definite thought, and that, consequently, there occurs the intensified feeling which we have already alluded to as a *rise in subject-consciousness*, with a *corresponding fall in object-consciousness.* This rise in the feeling element, and failure in the cognitive, leads readily to a delusional atate, as might be anticipated. This failure in the relational element of mind, due to some physical interruption of the molecular wave which traverses the connecting links between cell and cell, may pertain almost exclusively to certain of the higher evolved groups of feelings, and, so to speak, may (by a species of morbid dissection) issue in their gradual dissolution, while the other faculties remain more or less intact. The confusion of ideas, the delusional perversions, the indefiniteness of thought, accompanying the decay of the cognitive element, proceed *pari passu* with the rise in the feeling element (as exemplified in the monomania of pride, in the delusional states of general paralysis, and in the monomaniacal states of masturbatic insanity), and appear to indicate territorial implications of mind, possibly due to vascular changes. Here the question naturally arises how far this derangement is to be attributed to physical change within the nerve-fibre, or how far to interference by extraneous agencies, such as vasomotor changes, &c. The nervous wave which pervades the labyrinth of cell and fibre-network during active functionising, is normally determined thither by the act of attention; and, however we may explain this faculty (probably due to the establishment of a more rapid and vigorous circulation through the active area), its operation doubtless serves to intensify the intellectual or cognitive element of mind, and the relations between feeling and feeling become more pronounced and definite; or, in other words, thought is intensified. On the other hand, its *failure* not only reduces the nervous wave in volume and in vigour, but the relational lines cannot be forced thereby, and hence will ensue the states of mind already alluded to. Failure of attention is indubitably the earliest symptom in these states of partial exaltation. From this point of view, the *failure of attention* is the primary derangement, and the morbid result contrasts notably with that morbid entity engendered *ab extra.* If the bodily sensations (the cœnæsthesis) are seriously deranged; if altogether novel and obscure sensations are aroused by, *e.g.*, various visceral affections, it is notable that the personality may be endangered thereby, and a transformation of the *ego* may result. This fundamental change may be induced by the *persistent attention given to these morbid feelings* so aroused—feelings which embrace both the purely sensuous and cognitive elements—and overbalance previously-existing states, destroy the normal contrast, and become by this very faculty of attention intensified, and thus a morbid group of feelings is created and consolidated, which stands out in antagonism to the old self as a new *ego*. If sensory channels become the media for the transmission inwards to the sensorium of molecular waves of abnormal force, volume, or frequency, they will naturally arouse the sentinel faculty of attention to question their nature and relationship, and thus further aid the forcing of those relational lines which consolidate the whole. We have thus the possibility in the nervous mechanism of two morbid conditions arising, wholly distinct in character, whereby serious intellectual derangements may ensue. In old physiological parlance we may liken them to the *vis a fronte* and *vis a tergo* of circulatory phenomena; the former, represented by the faculty of attention, the latter, by the varying in-going currents along the sensory nerves. When the morbid transformation of the *cœnæsthesis* gives rise to the gradual pervading of the old by the new *ego* (*e.g.*, extreme egoism of puberty)—as the strengthening and elaborating, and integrating process advances under the auspices of a restricted attention—the definiteness of the delusional concept becomes notable, and no possibility of scepticism can arise; there is no excitement, but great mental calm.

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* *Principles of Psychology*, vol. ii., p. 578.

Hence, the essence of these forms of monomania is their constructive nature, whilst the distinctive feature of the perversions of the exalted senile dement is their dissolution; one is synthetic, the other analytic and destructive. Enfeeblement of the faculty of attention, therefore, evokes failure of the relational element of mind. Thought becomes obscured, and lessened in vigour and definiteness, the faculty of association of ideas is necessarily weakened; feelings are more crude, ill-defined, or poorly-demarcated from associated feelings; thought becomes less coherent, delusional concepts are apt to arise from exaggerated feelings, and loss of the contrasting and representative faculty, and excitement prevails. In the rapid reductions of general paralysis we have seen how all those feelings which are based upon the instincts of conservation of the self, or of the progeny (together with the complex and consolidated groups making up the social, religious and æsthetic sentiments) successively become deranged; and how in each the faculty of attention is first or earliest involved. This disease reproduces, in a terribly-rapid sequence, the morbid results which more gradually ensue in the decay of senile atrophy; the relational element or the nerve-cell prolongation is the earliest to succumb.

Senile Amnesia.—We have already seen that the earliest symptom apprising us of commencing decadence of mind in senility is the failure of memory for recent occurrences. The latest impressions reaching the sensorium may be so imperfectly registered as to be rapidly obliterated, or they may fail to establish the organic connections whereby they become more permanent constituents of the nervous mechanism. In the nexus of processes connected with, and extending around, a nerve-cell, we decipher the integration of structure upon which its permanence as a functional unit depends; and the more free such channels of communication become, the more fully organised is the structure, and the more stable and resistant to the encroachments of senile dissolutions. The latest requirements, however, are expressed in the structural modifications of the highest nervous arrangements where integration of structure is least advanced; and unlike those associations which have been called into activity over and over again, many thousand times, they fail in that nexus of communications necessary to their stability. Thus it becomes a matter of common observation that these senile subjects are oblivious to the most ordinary events occurring in their immediate presence; they will sit down and enjoy a hearty meal, and an hour after be unconscious of the fact; will meet a friend in converse, and forget immediately both the occurrence and the conversation. If the event be recalled, its connections with his recent experiences are so frail as to end in his relegating it to his bygone history. Ribot has well described the process of recollection and localisation of events in *time*. He indicates how the mind, as it were, travels back along certain definite lines, not recalling the myriad of events intervening, but leaping from certain prominent stations, or, as he calls them, "reference-points," until it arrives at the desired epoch, and thus, as it were, laying down a measuring-rod which defines the two points in *time*. "Without this abridged process and the disappearance of a prodigious number of terms, localisation in time would be very long and tedious, and restricted to very narrow limits"; and again, "by repetition the localisation becomes immediate, instantaneous, automatic" (pp. 52, 53). This does not accurately describe the process for all, inasmuch as the process varies much with different individuals. Thus, if we take into account those who "visualise" (in the sense in which this term is used by Galton), we find that the mind often recurs *immediately* to the event in question without apparent reference-points, and that the greater or less vividness or faintness of the image visualised tends to localise the event in time for such subjects; or that the mind travels forwards from a similar reference-point to the present, so reversing the process described by Ribot. This condition of mental, historical perspective steadily declines in senile dementia; so that localisation in *time* becomes to many quite impossible; present events fail to impress the organisation with their wonted vigour, and bygone experiences rise into undue prominence, rivalling in their more vivid reproduction the more recent occurrences, and become, as it were, projected into the present. This is what we might anticipate, as greater resistance is offered to the molecular currents of the highest nervous mechanism, the tide of nervous discharges must recede to the more deeply-organised structures. That the latest acquisitions and powers suffer earliest is also exemplified in the failure of memory for foreign languages, for late intellectual operations of abstruse nature, and, in fact, for all recent additions to the individual's knowledge; the more *specialised knowledge* always *fades soonest*. So proper-names are forgotten, a father may fail to recall his children by name, although he recognises each individually; or a patient, daily attended by nurse or doctor for years, may be unable to recall the name of either. Substantives are liable to be forgotten; as indicated by the hesitating speech, and usually a dead stop at a noun. Dr. Clouston truthfully portrays the typical senile speech as "a mixture of aphasic, amnesic, and paretic symptoms."* The decay of the intellectual is followed by failure of the sentient element of mind; emotion and feeling become slowly impaired, but at a much later date. Still, the patient may pursue his usual course of life, and all deeply-rooted sympathies, weaknesses, and prejudices may prevail in full force; the customary habits of a long life maintain their ascendancy; and though *special* aptitudes fail, the *more general* are still retained. Eventually, with their decline the subject is reduced to a mere vegetative state of existence. The whole process forcibly illustrates the law of dissolution, whereby the most-specialised, most-complex, and least-organised nervous arrangements suffer first, and the more-general, least-complex, and more-organised and stable nervous-arrangements are the last to succumb; in other words, the dissolution takes the course from the least to the most stable arrangements.

The following may be quoted as illustrative of the vivid hallucinations and terror seen in these cases of senile melancholia during the progress of atrophy, the dangerous impulsiveness to which they are liable, and the great trouble they give in our asylums.

M. M., aged eighty-five. A widow with two children; of Roman Catholic persuasion. Was admitted October 13th, 1886, under the following certificate—"Chatters about dead people whom she believes to be still living in an empty house close by; and one of whom she talks of marrying to-day. Believes herself to be possessed of property. Rambles about in search of imaginary objects. At one moment greatly distressed and crying, the next violent in speech and conduct. Has to be constantly watched to prevent her damaging windows and rambling from home. Threatens to kill her children and burn her house down; attempts to strike her grand-children with a poker." She was said to have been a very steady woman, with no insane or epileptic inheritance; one daughter had died of hæmoptysis, another of phthisis. It is affirmed that until five weeks ago she showed no mental derangement. She then became excitable, disorderly, tossed food down into the cellar for others to feed upon; soon became quite intractable, and several times rushed upon her daughter with a knife. Has been most destructive of clothing. "She has a large dolicho-cephalic head, a very long face, which is extremely wrinkled and pallid, and a fatuous expression. The hair is thin and white, jaws massive and prominent, few teeth remain. She is much emaciated. The superficial arteries show no very decided cording; heart's action feeble. There is slight pulmonary emphysema. Examination of the organs negative."

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* *Op. cit.*, p. 568.

Oct. 21st, 1886.—Restless and terrified last night; kept looking at, and pointing to, the windows, and declaring that "Mahony was coming to kill her."

Oct. 25th.—Very restless; incessantly trying to get out of the ward; asserts that she is "going home to-day." To have a mixture of tinctura hyoscyami with the double bromides of sodium and ammonium twice daily.

Nov. 8th.—Required feeding by the funnel; is deluded, unquiet, and speaks angrily against those who feed her. Talks much in unintelligible Irish brogue.

Dec. 23rd.—Asserts that "they are going to kill her; there are some knives with them there; there they are"—pointing to the ceiling. Is very agitated and restless; does not sleep well, and grows weaker.

Feb. 2nd, 1887.—Often excited and noisy; on the 30th ult. refused her dinner and remarked—"I won't take a bit; how do I know what poison I get?" At times fights with those who approach her, evidently in terror lest they should harm her. Is very feeble and thin; often requires a small dose of paraldehyd at night to secure sleep.

Premature Senility.—A careful comparative survey of the foregoing histories, with the case of *H. O.* detailed at p. 243, will at once convince us that we are dealing in both instances with two obviously-distinct affections of the nervous system. In the senile form we find the results of a general decline in the vital activities; the functions of nutrition, circulation, and the respiratory activities diminished. So also the blood-stream is impaired in quality, diminished in quantity, sluggish in its flow; and when we turn to the verdict of morbid anatomy, the blood-channels exhibit diseased tunics, narrowing of their lumen, tortuosity with attendant impairment of their resilience accompanying atheroma,—the tissue elements universally, and the brain-cells in particular, presenting fatty-change in their constituents, all indications of a senile retrogressive process.

The latter form (chronic cerebral atrophy) likewise presents a notable vascular change, but of a very different nature—a compensatory hypertrophy of the muscular tunic of the small vessels, especially emphasised in the cerebral arterioles, and very frequently associated with advanced atheroma of the large basal blood-vessels. This hypertrophic condition is also found in varying degrees in the kidneys, being identical with the changes described in the arterial system by Dr. George Johnson, as those of chronic Bright's disease, especially of that form

AMOUNT OF UREA EXCRETED DAILY IN CASES OF CHRONIC BRAIN ATROPHY, &c.

	Sex.	Age.	Urine Collected in Twenty-four Hours.		Specific Gravity.	Urea Estimated in Grains.	Empirical Estimate in Health. Parkes' Formula.	Body Weight in lbs. avoir.	Albumen.	DIET.
			Cubic Centim.	Ounces						
W. P., . Feb. 23, '88, .	M.	56	860	30	1,025	426·5	360	126	Nil.	Ordinary diet.
,, 24, ,, .			677	23	1,022	325·2			,,	
,, 26, ,, .			620	21	1,021	240·2			,,	
,, 27, ,, .			720	25	1,020	245·5			,,	
W. W., Feb., 24, '88, .	M.	59	2,680	94	1,010	290·7	417·8	178	,,	Mince-meat, milk, and farinaceous food.
,, 25, ,, .			1,820	64	1,009	282·1			,,	
,, 26, ,, .			1,100	38	1,012	238·7			,,	
,, 27, ,, .			1,010	35	1,010	183·6			,,	
,, 28, ,, .			1,620	57	1,011	276·3			,,	
Mar. 7, ,, .			1,750	61	1,012	122·0			,,	
,, 8, ,, .			700	24	1,018	238·7			,,	
,, 9, ,, .			1,030	36	1,012	239·4			,,	
,, 11, ,, .			670	23	1,019	186·9			,,	
J. M., . Mar. 7, '88, .	M.	54	900	31	1,016	251·1	383·6	143	,,	Essence of beef ; two pints of milk ; two eggs and rice pudding.
,, 8, ,, .			720	25	1,020	223·2			,,	
,, 9, ,, .			1,200	42	1,016	279·0			,,	
S. T., . Mar. 13, '88, .	M.	62	1,260	44	1,011	230·3	366·1	156	,,	Ordinary diet.
,, 14, ,, .			1,300	45	1,012	282·1			,,	
,, 16, ,, .			1,130	39	1,015	262·7			,,	
,, 17, ,, .			1,000	35	1,015	232·5			,,	
,, 18, ,, .			720	25	1,018	200·8			,,	

AMOUNT OF UREA EXCRETED DAILY IN CASES OF CHRONIC BRAIN ATROPHY, &c.—*Continued.*

	Sex.	Age.	Urine Collected in Twenty-four Hours		Specific Gravity.	Urea Estimated in Grains.	Empirical Estimate in Health. Parkes' Formula.	Body Weight in lbs. avoir.	Albumen.	DIET.
			Cubic Centim.	Ounces.						
J. R. S., Mar. 20, '88, .	M.	53	1,030	36	1,013	207·5	587·3	208	Nil.	Ordinary diet.
E. L., . Feb. 21, '88, .	F.	47	1,010	35	1,010	187·8	230·3	96	Slightly turbid.	Meat dinner, and two pints of milk and farinaceous food.
,, 22, ,, .			1,650	57	1,010	255·7			Flocculent.	
,, 23, ,, .			1,130	39	1,010	175·0			,,	
S. W., . Mar. 17, '88, .	F.	46	1,610	56	1,014	349·3	240	100	Deposit $\frac{1}{16}$th.	Two pints of milk, one egg, arrowroot, oatmeal gruel (cod liver oil).
,, 18, ,, .			1,260	44	1,015	263·5			,,	
,, 19, ,, .			1,060	37	1,016	262·8			,,	
,, 20, ,, .			780	27	1,017	253·8			,,	
J. H., . Mar. 23, '88, .	M.	39	2,280	79	1,014	494·7	435·9	130	Nil.	Ordinary diet.
,, 24, ,, .			1,130	39	1,018	350·3			,,	
,, 25, ,, .			1,360	47	1,015	421·6			,,	
,, 27, ,, .			1,886	65	1,015	541·5			,,	
T. R., . Mar. 3, '88, .	M.	40	1,180	41	1,023	621·8	557·7	158	,,	Essence of beef, 2 pints milk, two eggs.
,, 4, ,, .			1,350	47	1,024	711·4			,,	
,, 5, ,, .			1,185	38	1,027	752·9			,,	
,, 7, ,, .			860	30	1,026	599·8			,,	
,, 8, ,, .			1,040	36	1,022	693·1			,,	

associated with the contracted kidney. The imperfect renal function is manifested in nearly all these instances of chronic brain atrophy, as may be illustrated in the foregoing table, containing the results of examination of the urine in seven typical instances, followed for the contrast by a case of general paralysis (*J. H.*), and one of acute delirious mania (*T. R.*). The urea was estimated in these cases by means of Gerrard's ureometer, and the amount in health normal for each individual was obtained by Parkes' empirical formula, having regard to the sex, body-weight, quiescent, or active condition of the patient. When these conditions, together with those dependent upon diet are allowed for, we still find a considerable deficit in the amount of urea daily eliminated. Two cases out of the seven revealed albumen, and only to a slight degree.

The history of such patients points very decisively towards the morbid change being expressive of a general constitutional derangement, which we must identify with chronic Bright's disease, and which expends its force with varying degrees of intensity—

1. Upon the minute arterioles of the kidney, productive of the small contracted organ met with—
2. Upon the minute cerebral arterioles issuing in this chronic atrophy of the brain—
3. Or is chiefly emphasised in the vascular apparatus of the spinal cord.

In all such cases alike we have similarly-disturbed functional prodromata with associated neuroses; in all the high-tension pulse is a characteristic indication; in all a similar effect is reproduced upon the heart and large blood-vessels.

If we contrast the senile pulse where atheromatous degeneration prevails with that of a typical instance of chronic brain atrophy, we find the former presents a tortuous artery, unduly prominent and visibly mobile with each pulsation, giving a sphygmogram of exceedingly low tension, a vertical percussion up-stroke, an almost equally sudden collapse, and a frequent diminution of the dicrotic wave varying with the endo-cardial and aortic conditions. There may possibly be a degree of *aortitis deformans*, with loss of normal elasticity, associated with more or less aortic regurgitation; secondary undulations during diastole are usually absent.

In these cases of cerebral Bright's disease, however, the pulse is small, very hard and incompressible, in fact, indicative of a high arterial tension, modified more or less by a hypertrophied left ventricle; there are also a well-marked percussion-impulse varying with the degrees of hypertrophy, a well-sustained tidal and good dicrotic wave, often succeeded by a slight secondary undulation during the diastolic pause (arterial elasticity). In the case of extensive atheromatous disease a tracing-pressure of from 30 to 60 grammes suffices, and the occlusion-pressure is very low; in the pulse of the subject of chronic brain atrophy the hardness is so marked that we have often to employ a pressure of 180 grammes, and with a large hypertrophied heart the pressure often has to be still further increased.

The obstruction in all these cases is due to the hypertrophy of the *tunica muscularis*. As was indicated years since by Dr. Broadbent,* the peculiar character of the sphygmogram is immediately altered by the exhibition of amyl, and also by the onset of pyrexia. With the atheromatous condition, however, the exhibition of amyl, of alcohol, and especially of alcohol subsequent to chloral, produces such paralysis of the muscular tunic as often to result in a dangerous vaso-motor relaxation—a condition not seen in chronic brain atrophy except with the association of extensive atheroma.

As in the early stage of Bright's disease the chief symptoms are those referrible to disordered digestive and assimilative functions—pyrosis, eructations, gastralgia, nausea, loathing of food, or a capricious appetite may long prevail and constitute the premonitory note of alarm. Then follow evidences of depraved blood; of the hydræmia associated with adventitious irritating substances which fail to be eliminated; the red-corpuscles decrease in number, and the circulatory energy becomes impaired. Yet it is in the early prominence of nervous symptoms that we see indicated the tendency to issue in brain disease. All cases of Bright's disease exhibit more or less of these nervous derangements, but such symptoms are peculiarly emphasised in the cases with which we are now concerned. Headache (often hemicrania), giddiness, vertiginous seizures, syncopal attacks, severe palpitation, disordered sensation (special and general) are peculiarly prominent. Some subjects are victims to severe forms of facial neuralgia, others complain of great mental torpor with somnolence or even stupor. A deranged state of the blood, as regards its quality and its supply to the central nervous system, explains to a large extent these symptoms of disordered innervation.

Just as with the renal implication of chronic Bright's disease, the intensity of constitutional and local indications may bear no constant relationship to each other—so in these cases the degree to which the nervous-centres are involved will greatly vary for each individual case, the local having no constant relationship to the constitutional. With the implication of the nervous-centres we may find associated every degree of renal and hepatic degeneration and cardiac hypertrophy; but it is more usual to find the kidneys in a state of early interstitial fibrosis than very extensively affected; we usually find one organ in a much more advanced state of degeneration than the other.

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* Discussion at the Royal Medical and Chirurgical Society, *Med. Times and Gaz.*, Dec., 1872.

The special determining factor upon which depends this tendency to implication of the cerebrum in particular, in cases of chronic brain atrophy, would appear from our statistics to be alcohol; a large percentage of alcoholic cases undoubtedly succumb to this affection. The selective power of the brain for alcohol has probably much to do with this determination of morbid activity towards the cerebral blood-vessels. Recognising in most, if not in all, such cases, the injurious effect of alcohol upon the blood-plasma, we cannot too strongly insist upon the importance of defining the constitutional as apart from the local derangement in the earliest stages of this affection, and of at once treating such conditions with the object of evading the local implication.

THE TREATMENT OF INSANITY.

Contents.—Physiological Element in Treatment—The Moral Element—The Mental Nurse—Individualised Treatment—Hospitals for the Acute Insane—Modern Revolution in Treatment—Rest and Exercise—Treatment of Delusion; Destructive Habits; Suicidal Tendencies—Therapeutics—*Rôle* of the more Important Sedatives Employed — Chloral — Chloralamide — Paraldehyd — Sulphonal — Trional and Tetronal—Hyoscyamine—Duboisine—Opium—Cannabis Indica—Conium.

The treatment of insanity naturally resolves itself into (*a*) Physiological or Hygienic; (*b*) Moral; and (*c*) Therapeutic Measures.

It is necessary that we appreciate the implication of these terms, just as we must recognise that a strict classification under these headings cannot be always logically claimed for any special line of treatment adopted; thus, the physiological often includes a moral element, the moral often has a notably physiological operation, whilst the therapeutic again has, under certain conditions, an equally strong moral effect.

The Physiological Element in Treatment.—By these measures we seek either to convey the organism into a more suitable environment, or so to modify the existing environment as to reduce to a minimum the friction established betwixt the two; in other words, to favour, by the removal of certain ascertained inimical factors, the healthy and normal adaptation of the organism to its environment. In every sense humanitarian principles guide us here, and the law of the survival of the fittest is recognised, only to be opposed and combatted by the full force of modern altruistic measures.

All sources of irritation to the organism, where detected, must be removed, whether these be physical, mental, or both conjoined.

Exhausting occupations, mental strain, especially with worry and coincident anxiety, irregular habits of life, and local associations which are recognised as tending to foster a feeling of antagonism to the environment; all these should be met and combatted, and this, as is well known, can, in by far the larger number of cases, only be ensured by the removal of the patient to fresh surroundings, by placing him where the conditions of life are simpler, and where hygienic considerations are stringently kept in view.

Just as we strive to remove all sources of irritation, so we endeavour to eliminate such noxious agencies as by self indulgence, or by the influence of injudicious friends, have entered into the vicious circle of his life—alcoholic indulgence, the morphia, chloral, or cocaine habit, sexual excess, morbid excitement of any character, solitary habits, and morbid introspection. Wherever the organism is exposed to peril, as in suicidal conditions, extreme violence, absolute refusal of food, long continued and complete sleeplessness, the physiological factor in treatment plays a conspicuous *rôle*, since the removal of the patient to an asylum or hospital is then almost invariably demanded, if only to secure the full moral and therapeutic effect of treatment.

The Moral Element in Treatment.—By moral treatment we refer exclusively to the direct effect of mind upon mind, whether this be of the nature of a soothing, calmative influence, or of judicious repressive measures adopted towards any vicious or dangerous tendencies upon the part of the patient; in other words, the psychical environment is so modified as to induce a healthy response, and encourage normal adaptation. The physician here largely depends upon the nurse; the constant companionship of a suitable attendant is the sheet-anchor of success. Here it is, also, that a strong contrast can be drawn between the usual hospital and mental nurse. We must admit that the intelligent, responsive, and successful mental nurse has a far more complicated sphere of labour to engage in than the nurse in the medical and surgical wards of a hospital.

(*a*) *The Mental Nurse.*—It is not so generally recognised by those who interest themselves in the furtherance of nursing schemes, that, whilst the hospital nurse is usually incapable of taking charge of mental cases from lack of experience in so special a department, the asylum nurse, on the other hand, if well trained, is fitted for hospital work, whilst her experience in mental nursing makes her a most invaluable adjunct to the usual staff.* The nurse who can enter

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* There is a sad lack of the co-operative spirit betwixt these two classes of Institutions, strange as it may appear, and, perhaps, not the least important obstacle to a reform in this direction lies in the absolutely untruthful suggestion so freely indulged in by those who should know better—viz., that the asylum nurse is a raw recruit untrained for hospital work and not certificated. The establishment

LANE [illegible]

most closely into the mind of her patient, who can probe her feelings with instinctive readiness, and adapt her resources to the varying moods presented, is, indeed, a valuable auxiliary to the medical attendant. A quiet, calm, gentle demeanour, free from the least flurry, a constant cheerfulness and brightness unruffled by any of her patient's vagaries, an absolutely even temper, great patience and forbearance, are indispensable requisites. The nurse should be self-possessed, but not self-assertive; should exhibit a firmness united with gentleness and the quiet self-reliance begot alone of a ripe experience, and a complete faith in the virtue and efficacy of patience conjoined with sympathy. A watchfulness ever wary, yet inobtrusive; a studious regard to all her patient's susceptibilities—nervousness, irritability, moroseness, despondency—meeting every varying phase with its suitable corrective. This is a function demanding the highest social qualifications a nurse could be required to exhibit, and, as might be expected, the woman in this capacity is far more successful than the male attendant. These, then, are some of the qualifications demanded of a good mental nurse, and if such be required of a nurse in charge of a *single case*, how much more complicated becomes her *rôle* when placed in charge of a large ward, where she also has to encounter and control the actions and interactions between a number of patients all varying more or less in the special treatment applicable to each case? It is in this capacity that her tact and address will be best displayed, as she has not only to keep a watchful supervision over her patient's welfare, but also to control and train a subordinate staff of nurses, and to strive by her influence to spread a spirit of loyalty and discipline amongst the junior nurses.

The conscientious discharge of such functions entails a heavy burden of responsibility, demands a healthy physique to stand the strain, and involves upon those responsible for the employment of such nurses the duty of providing them with adequate rest, recreation, and well-regulated and substantial dietary.

(*b*) *Individualised Treatment.*—The whole aim of moral treatment is that of individualising the subject to the fullest possible extent; each case must be treated upon its own merits, to obtain the best results; each patient must receive as much attention as the physician and nurse can bestow. Here it is that a great stumbling-block presents itself to the alienist in charge of our large asylums, where the vast accumulation of the chronic insane paralyse his best efforts. The large proportion of

by the Medico-Psychological Association of examinations for certificates in mental nursing, and the ambulance and nursing classes held now in all British asylums, is of course a sufficient refutation to this most unjust aspersion. That the public and the profession fully appreciate the higher functions of the mental nurse is indicated by the higher scale asked for her services and so readily granted.

the chronic and incurable class in our asylums require no such elaborate system as is provided necessarily for the minority—the recent and acute. Attempts have been made to obtaining redress for this condition of things at the hands of the Union authorities, it being suggested that large numbers of the incurable and harmless might, with wisdom, be relegated to the large union infirmaries, which, if somewhat better officered, might, at a much reduced rate, support the great bulk of the chronic residue. The attempt has, however, not met with encouragement, and it is more than probable that our Councils will eventually solve the question by building asylums upon more economical principles for this class of the insane; and by erecting hospitals for the acute and recent in connection with each large county asylum; this appears to be the more rational solution to the problem. The hospital should be built upon the most approved scientific principles, and should embrace an administrative block capable of meeting the fullest demands of modern treatment. It should afford facilities in the form of laboratories for scientific research at the hands of the medical staff; and should aim at being a centre for the teaching of psychiatric medicine, since our asylums alone embrace the *material* for such studies. Into this hospital should be received all incoming cases of insanity, those recognised as chronic and incurable being at once drafted off to the larger institution, and those alone retained here who present hopes of final recovery or such relief as is compatible with discharge to their homes. No distractions would thus occur to the medical and nursing staff by the multitudinous and importunate demands of the chronic class, which, at present, so largely enslave their attention and limit their utility for the real work of asylum life. All who have had any practical acquaintance with the insane must recall cases where the difficulty of segregating, so as to remove certain acute cases from the influence of irritating chronic patients, retarded recovery; and where the want of more individualised attention appeared answerable for the gradual drifting of such cases into secondary dementia; nor is it a rare occasion to see a notable change for the better effected in such cases when transferred to other wards, where the sources of annoyance were fewer, and where the nurse was able to afford more continuous attention, or was better adapted for her duties.

Therapeutic Measures.—During the past quarter of a century the treatment of insanity by therapeutic measures has undergone a complete revolution. Three decades ago antiphlogistic theories still maintained their ascendancy over certain minds, and depletion by bleeding, either lancet or leech, active purgation, powerful drugs, especially the tartarated antimony, were all in vogue. Excited patients were often kept for days together on the verge of narcosis, croton oil and tartarated antimonial ointments were vigorously ru into the scalp, and this was considered an heroic and consistent of treatment. All this is now altered; not so much, perhaps, b demonstration of the inutility or even actively injurious resul these procedures, as by our growing acquaintance with the stru and functions of the nervous system, its physiological chemistry pathology. And thus, as with the death of all old systems, a hea scepticism of such treatment arose, not by any critical exposure vicious nature, but by the general advance in various departmen knowledge.

The reaction against such heroic measures had, as its fundame conception, the nature of insanity as a *reduction* both in physical in mental vigour; and, as a necessary corollary, the employmen means to *build up* an exhausted nervous system, and above all th to avoid lowering the vital energies of the fabric. Yet, as in all reactions, the sceptical spirit often appears to have carried some votaries too far, their faith in *all* medicaments being so violated they are even prepared to affirm that all alike are useless. On other hand, old systems and habits of thought die hard, and, even we have faint revivals of these old-fashioned views afforded us, if to so serious a degree, at least to an extent that is often ope censure; thus we still hear rest in bed strongly deprecated for of excitement, although it is almost universally admitted by the experienced as a powerful agent for the reduction of excitement the renovation of the nervous system. And thus we find at present time extreme views held still by a few, the more rational of treatment lying, as is usual, in the golden mean.

Rest and Exercise.—A very large proportion of the patients signed to our asylums in the early days of treatment imperati demand rest in every form in which it can be enjoined. To pla recent case of acute excitement amongst a number of others simil noisy and boisterous would be the height of absurdity and thought ness; just as it would be to insist upon the attendance at a da theatrical entertainment, or concert of a patient plunged into abject misery of melancholia. For all *recent* cases rest in bed is o our best adjuvants to treatment; the sleeplessness, the irritatio removal to an asylum, and the various unfavourable circumstance which the patient has been exposed call for immediate rest, soot influences, quiet and even a darkened room. All recent cases o acute character are wisely retained in bed for at least two days admission; we invariably, if practicable, make a careful examina into the mental and bodily condition on the second day. All c sufficiently conscious are thus brought to feel that they are expose a rational system of treatment. It is then decided whether fur rest in bed through the day be desirable or not, and, to a large extent, the reply depends upon the patient's physical condition. In a large number it will be found advisable to keep them in bed for at least a part of the day for some little time; and to encourage meanwhile the reparative processes which have probably been much neglected prior to their arrival. Debilitated and senile subjects, maniacal or melancholic, refusing food persistently will thus often imperatively demand rest in bed throughout the day; and so the turbulence of acute excitement in exhausted subjects is far better treated in the quiet of a side room than exposed to the irritation of other cases in a day-room. In such cases, to enjoin out-door exercise indiscriminately might occasion much harm and would certainly prejudice the recovery unfavourably.

In the large Berlin State asylums this line of treatment is followed; and especially at Dalldorf, which, to our mind, reflects the most advanced and rational aspects of modern psychiatric medicine in Germany, it was pleasing to note the genuine hospital character of the treatment adopted. Dr. Koenig of that institution kindly favoured us with his views on rest as an agent employed by himself and other alienists in the early days of mental disease. He informs us that all recent cases are kept in bed after admission for *twenty-four hours at least* with the object of calming excitement, impressing the patient with the *hospital* nature of the treatment adopted. On the second day, some are allowed to leave their bed; others are kept at rest therein for weeks or even months unless they resent the treatment, when it is modified in accordance with their wishes. Dr. Koenig insists upon the necessity of adequate out-door exercise where this can be borne; but he by no means intends to indicate its indiscriminate application to all cases of excitement; his plea is one of moderation, and he strongly repudiates extremes in either method of treatment. We agree with him that it is often of service to take certain cases of recent excitement out for an hour's walk, subsequently allowing them to return to bed rather than to remain up among their fellow patients.

It is, we think, quite wrong to adopt any absolute rule for the exercise and rest of recent cases; each case must be treated on its own merits, and the individualised treatment nowhere indicates its value so emphatically as in its application to the exercise, recreation, employment, and rest afforded to recent cases of insanity.

That the surplus energy of the maniac should be carried off by severe muscular exercise is a doctrine which, in recent cases at least, may be carried to a very dangerous extent. In fact, the explosiveness of recent mania and melancholia, as due to cortical instability, and the withdrawal of higher control indicates a demand for the *conservation* of energy so as to lead it off by channels directly subservient to the use of the organism; and by quiet, by nourishing food, tonic regimen, and complete physiological rest to improve the nerve tone and central stability.

Another fallacy which is introduced by extreme views upon exercise in recent insanity is that the boisterousness, noise, and strugglings of the acute maniac serve so far to demoralise the orderly discipline of a ward and the domestic comfort of the inmates, that the resort to sedatives is likely to be adopted, where, otherwise, they would not be given; in this case, the sedative becomes a form of chemical restraint and is much to be deprecated; infinitely preferable is it that the patient should be isolated under such conditions, than that we should be driven in recent cases to employ sedatives for such a purpose.

A difficulty also arises from the accumulation in our wards of the excited chronic class, some of whom are at times utterly unfit for associating with the recent and acute inmate. According to some extremists, these patients, hopelessly insane as they are, should be kept in association with others, however excited and noisy or violent they may be, rather than be isolated for their prolonged paroxysms of excitement. To this doctrine we can never subscribe; fully recognising as we do the vicious nature of isolation carried to an undue extent, we must distinguish between the hopelessly incurable and the recent case; and we assert that the injury thus inflicted upon recent cases by the irritation of such associates is incalculably greater than that arising from any temporary isolation of the chronic class during their most protracted outbursts of mania.

Amongst chronic cases, it is true, excitement may often be met by employment and vigorous exercise; but, even here, we must differentiate between those who suffer and those who do not suffer from physical disability, from cardiac affections, or serious cardio-vascular degenerations so frequent amongst these cases.

Delusions.—It is a fully recognised principle that delusive beliefs and utterances are never to be met with directly adverse criticism, and on no account is the physician or the nurse to oppose them by hostile argument, but rather to ignore their existence, or at most to refuse quietly but firmly to admit the delusive statements made. The attention of melancholic patients tend to be far too engrossed by their delusive beliefs and hallucinations to permit us to further emphasise these states by encouraging discussion on the subject. The primary object of the nurse should be to divert the attention from all such themes, and to employ the energies in directions least likely to foster self-engrossment, and least apt to recall morbid associations.

In this way we often find that association with other patients does far more benefit than the isolation necessitated by a single charge. The thoughtful nurse will encourage all that tends to distract the attention from any painful theme by directly encouraging a healthy interest in their fellow patients, their assistance being sought for wherever it can be legitimately accorded. To the social nexus established in this way under the comparatively novel circumstances of asylum life in our wards, is to be attributed much of the improvement which occurs in cases of melancholic depression and delusional perversion. In eliciting the delusions of the insane for clinical purposes, it is, therefore, wise to forego frequent discussion on the subject, and these remarks apply especially to such delusions as tend to become fixed, and those which show an early tendency towards systematisation—*e.g.*, adolescent insanity, the melancholic delusions of the climacteric period, and those of alcoholic insanity.

In the former, solitary and vicious habits must especially be prevented, and wherever possible, association both night and day is to be strictly enjoined with the object of correcting or suppressing such habits, which lead most certainly to hallucinatory conditions with all their persistency and far-reaching consequences.

If, however, we recognise the primary systematised form of insanity as the evolved psychosis, it matters little whether the delusions be the subject theme of conversation or not, the delusional perversion will steadily, but surely, progress to a hopeless termination.

Destructive Habits.—Destructiveness in the insane may be the outcome of pure wanton mischief, a love of annoying others, or the act of a passionate outburst, such as is so frequently seen in the epileptic or congenital imbecile; on the other hand, it is very often the outcome of maniacal reductions, indicating the outflow of superabundant energy, and so, likewise, is the restless excitement of dementia, whether senile or paralytic; lastly, it may be the result of sudden incontrollable impulse after epileptic and alcoholic reductions, and especially in the insanity of early childhood. Thus the dementia of general paralysis is frequently accompanied by the destruction of clothing and bedding during paroxysms of excitement; epileptic furor, following upon epileptic seizures, is often accompanied by extreme destructiveness and other dangerous symptoms of a purely impulsive nature. As illustrative of the destructive *impulse* may be quoted the case of an adult male, a criminal lunatic, who would, ever and anon, be found in his cell stark naked, with all his clothing torn to shreds at his feet. On being questioned as to his motive, with a silly half-vacant smile he would reply, "I don't know; I suppose I *must* have done it." He was soon found to have had an attack of petit mal, and, subsequently to his removal to an asylum, he would, without any apparent cause, suddenly turn round and strike anyone near him full in the face. When admonished, he would affirm with a complacent smile that he could not help doing so. His condition remained uncorrected after several months' treatment, both moral and therapeutic, and was purel the outcome of the epileptic neurosis.

The incorrigible destructiveness of chronic mania is also a source o much annoyance and outlay in our large asylums.

When we are dealing with mischievous and passionate outbursts moral treatment is a powerful adjunct; the threat to suspend al indulgencies will alone suffice in many cases to arrest such tendencies The change to another ward, with the moral effect of new companions may effect the same result, and the isolation in bed with none bu indestructible clothing will prove sufficiently deterrent in other cases In the destructive tendencies of maniacal excitement on the other hand moral means are of little avail. The constant presence of the nurse her firm and repeated interference with such habits may be of avail i some cases, but in the majority it is found absolutely essential t supply the patient with strong indestructible dress material an bedding, including well-padded ticking rugs, whilst the clothing mus be so secured as not to be readily removed or loosened by the patient Locked boots are also essential for these cases occasionally. Th impulsive forms of destructiveness such as occur in epilepsy an alcoholism will likewise derive but little benefit from moral treatment extreme vigilance will have to be exercised, whilst the treatmen proper to the convulsive neuroses is being furthered.

A form of destructiveness, found not unfrequently in melancholi states, and also in certain cases of dementia, is that of pulling out th hair from the head till patches absolutely bare are found, or the whol scalp is left bald. Some patients will pluck out hair by hair, and, i not carefully watched, will swallow such; large accummulations hav thus occurred in the stomach or intestinal tract. Idiots will occa sionally do this; and we recall such a case where it was carried on s stealthily at long intervals that a large mass of hair, agglutinated b mucus, became impacted at the pyloric orifice of the stomach, and le to a fatal result.* In such cases we have no better resource, upon th failure of moral suasion, than to adopt restraint, which is here full justifiable.

In chronic melancholia, again, we often find the querulous di content express itself in such destructiveness; the picking out threads, the tearing up of seams in the clothing, or the ripping ope of the bedclothes. Here much can be done by the soothing influen of the nurse, who will endeavour at the same time to repress suc habits by distracting the attention into other directions. When th

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*A similar case is recorded fully by Dr. Cobbold. The patient was an epilept diot, and the difficulties of detection, diagnosis, and the post-mortem conditio were almost identical with our own case. *Journal Mental Science*, April, 188 p. 52.

symptom is the outcome of the abnormal flow of energy, as in most cases of excitement, active muscular exercise out of doors, under the charge of one or two nurses, will do much to arrest the tendency; excitement will thereby be often much allayed, and on the return of the patient to the quiet of her room, a refreshing sleep may result.

Suicidal Tendency.—This tendency manifests itself in such varied forms, and the means sought for the accomplishment of the act are so diverse, that it is not to be wondered at that our efforts are thwarted at times, despite the facilities which a large asylum usually commands for the observation and control of the subject. The negative feeling prompting to suicide need be by no means profound; in fact, it may only occasionally assert itself as a sort of slight intensification of the prevailing depression; or the subject may affirm that, when the feeling arises, he has not the nerve to accomplish the act. It may be constantly present and carefully concealed from the medical attendant, or it may prove only too evident in the fixed, hopeless and painful expression, and the furtive glance, and the incessant restlessness, which are only too well recognised by the experienced observer. It may display itself by determined and constant plotting to accomplish its object, with critical periods of desperate efforts; or, again, it may prove purely convulsive and paroxysmal in nature, following upon an epileptic seizure, or during the "horrors" of delirium tremens. It may assert itself in long-continued refusal of food, violent resistance to forcible feeding, kept up, it may be, for weeks and months together, and often with attempts at inducing regurgitation after forcible feeding. It will be readily admitted that under these varied phases the treatment must be adapted to the individual case. It might be thought that, in the majority of cases of determined suicidal tendency, moral influences would prove of little avail. As a fact, however, this is not the case, and experience teaches us how wonderfully beneficial may be the influence emanating from a cheerful, sympathetic, firm, and efficient nurse. The rapid change which often takes place in these melancholic patients, when removed from inimical influences to the care of a nurse endued with tact and discrimination, is one of the most surprising and satisfactory experiences of asylum treatment.

It has been stated by Morselli, and noted by others, that certain classes of the population adopt almost invariably the same suicidal methods. It is so also in our insane community; certain individuals always choose by preference the same methods for accomplishing the deed; hence it becomes essential to learn, as far as possible, the former tendencies of our case, and for the nurse to observe carefully what are the special morbid proclivities of her charge. In all cases alike, it will be well to remove at once all possible sources of peril—*e.g.*, scissors, knives, needles, cord, matches, &c.; it may be necessary to remove false teeth, and in desperate cases to cut the hair short, or remove anything that may be used as a ligature. The windows must be secured, so that no danger from precipitation need be feared; the open fire-places may need a guard, but the constant presence of the nurse must be chiefly relied upon to secure the patient from the risk of her impulses. It should always be remembered by the nurse that her sympathy may be misdirected by constant reference to her patient's painful mental states; as in delusional states, the depression must be more or less ignored, combatted by a constant cheerfulness, every effort being made to induce an initiative on the part of the patient. We have thus found good results accrue by affording such subjects the means of inviting and entertaining patients from other wards, and playing the *rôle* of hostess for the nonce; any such device for distracting the attention from the prevailing egoism must prove beneficial, and every sign of a returning initiative may be welcomed as of good omen. Despite the close observation thus enjoined, it must invariably be the rule that the restriction of individual liberty should be as inobtrusive as is compatible with safety; and as soon as good reason is afforded us for assuming that the suicidal feeling has lost its hold, we should not neglect to indicate to our charge our returning confidence. Unnecessary restrictions too long prolonged are injurious, in so far that they both irritate and tend to destroy the growing self-respect and assurance. With respect to entertainments carried out on an extensive sale, our experience is not altogether favourable; for the chronic residents of a large asylum the frequent dances, concerts, theatrical entertainments may serve well to relieve the *tedium vitæ*; but as an auxiliary in the treatment of acute insanity, such means must play a very secondary part in the furtherance of our cures. If their *rôle* here be considered fairly, we should avow that in many cases of depression they are decidedly injurious when indiscriminately employed; that they often impose more suffering upon the subject; and that, in most instances, they are more beneficial in the later stages, when returning interest in their surroundings indicates that convalescence has set in. We are strongly of opinion that we cannot too carefully individualise and select our subjects when the question of entertainment comes to the fore. In like manner, the wise physician will very carefully consider the advisability of permitting attendance on religious services, and cautiously note their effect when they are permitted.

Treatment of Depression.—The diet should be nutritious and unstimulating; alcohol is unnecessary in most cases of mental disease, and positively injurious in explosive states and in epileptic insanity in particular. In all forms where diminished control results in morbid impulse, of the nature of obsessions, alcohol is practically contra-indicated. It is of value where cardiac energy is greatly impaired and exhaustion imminent, as *e.g.*, in persistent refusal of food, but even here it should be given in graduated doses according to the reaction of each individual case; it is of value where depressant drugs, such as chloral, have to be administered despite a failing heart and impaired circulation. In long continued melancholic agitation, with sleeplessness and emaciation, its use is often imperatively demanded; in such cases, no one can dissent from Lauder Brunton's view that it is a *food* in a certain sense; it economises the proteid metabolism. With the same object in view, we should insist on a due supply of carbohydrates in the food, as tending to restrict proteid transformation. In simple cases of melancholia, where there is little tendency to tissue waste, our attention will be chiefly directed to maintaining the appetite, relieving torpor of liver and bowel, and ensuring that the digestive and assimilative functions be duly performed; whilst we add to the dietary such elements as are obviously indicated by the patient's condition, such as iron, arsenic, the compound phosphates, cod-liver oil emulsion, and malt extracts.

It is worse than useless to give our subject a surplus of nitrogenous food over and above his capacity for assimilating it; we dare not violate physiological rules so far, otherwise we load the bowel with decomposing food and add to our patient's misery. In fact, by so doing we disorder nutrition at its fountain head, and render auto-intoxication a very serious probability. An abundant milk supply should be our staple article of diet; fish, meat, eggs should be given liberally, with a varied vegetable diet at regular intervals and with discretion. Where resistance to food occurs, compulsory feeding must be enjoined, but only after every effort has been made by tact and kindly persuasion to induce the patient to take food voluntarily. Obstinate refusal must be met with determination and vigour; three meals a day should be given forcibly, and, in very exhausted states, a fourth feed may with advantage be given late at night. It is by no means unusual for deluded subjects to receive willingly from the medical attendant or night nurse what the day attendant failed to administer; and, occasionally, a patient will take by stealth what she obstinately refuses when offered to her. The question of removal from home of mild forms of depression will depend chiefly upon the social circumstances of the family; if the means are forthcoming, a private nurse may be engaged with advantage, but, even then, association with the friends is not admissible. This is one reason why residence away from home or travelling in single charge is often essential. Even with suicidal tendencies, single charge, under a thoroughly competent nurse, is not negatived.

Depression at Puberty.—Hysterical symptoms in pubescent girls often assume a suicidal feature; usually the feeling is obtrusively emphasised; there is a morbid plea for sympathy which is by no means necessarily genuine; removal to an asylum under these circumstances would often be felt as a lasting disgrace, and extreme delicacy must dictate to the medical attendant the proper course to pursue. Travelling in single charge or residence away from friends may be enjoined here with advantage; but, it will at once be seen that the poorer class cannot afford the cost which this implies, and must fall back upon asylum treatment as the only resource. In such cases, the tact and ability of the nurse will be tried to the uttermost.

Neurasthenia and Depression.—In the case of certain neurasthenic subjects, the ordinary social obligations and conventionalities of life become not only burdensome, but a source of constantly increasing irritation; it is eminently unwise to insist upon these functions under such circumstances; much harm may be done thereby, and great risk run. The relatives will often strive to induce the sufferer to overcome his scruples by insisting upon attendance at religious services, public amusements, or even by the performance of public functions, despite the fact that he regards it in the light of mental torture; such a stoical doctrine is, to say the least, irrational. A wise attendant will at once remove all such sources of annoyance, will lead the mind into other and more congenial channels of thought and action, and, only when returning health demonstrates increasing self-assurance, will it be wise to suggest a return to former conditions of life; even then the suggestion should be so framed as to appear to be the patient's own desire.

Melancholy with Stupor.—Apart from the general principles of treatment above noted, these cases will derive much advantage from the administration of baths—electric, shower, and spinal douche; whilst masso-therapeutics, shampooing, with the application of the constant current to the scalp may be conjoined with advantage. These cases, of course, demand great care and watchfulness to secure a due amount of nourishment being taken, and to ensure the regular action of the bowel and the emptying of the bladder.

In all cases of depression we endeavour, if possible, to secure *natural* sleep before resorting to sedatives; but delay is dangerous in **acute melancholia,** and the symptoms are grave from the outset; the persistent refusal of food, the absolute insomnia, and acute depression, entail such rapid wasting and exhaustion that we cannot be too prompt in securing sleep even at the cost of sedative treatment. Food must be given regularly, and, if necessary, with force; and, as in the case of acute delirious mania, the patient must be treated from the first as an invalid in a perilous condition. The removal of all sources of irritation, absolute quiet, a darkened room, free ventilation of the sick room, a cool equable temperature, cleanliness, order, a quiet, firm, and cheerful demeanour on the part of the nurse, will all tend to influence the patient's mind, and to induce the natural repose required.

The depression occurring at the *climacteric* and during *lactation*, if of a mild character, may often be treated at home, or away from home with a suitable nurse; should suicidal promptings be strong, however, removal to an asylum will be most judicious.

Treatment of Excitement.—Most cases of excitement are still more unsuitable for home nursing than is the case with depression; mild hysterical forms and very early outbreaks of senile insanity, as also puerperal and gestational forms, should all have the benefit of home treatment when this can be conjoined with good nursing and safety. The dangerous impulsiveness of puerperal subjects render them the more anxious class to deal with in this way; yet it is peculiarly desirable that a trial should be made ere resorting to the severe procedure of asylum supervision.

Pubescent and Adolescent Insanity.—It is wise to resort to early removal from home in these subjects; apart from the necessity of breaking through associations, which often tend towards fostering systematised delusions, we have to shield the patient from the results of his own vicious habits, and from the dangerous impulsiveness to which he exposes himself and his friends. In this form exercise and employment are of vast importance, as is the case with all recurrent seizures of youthful subjects; they aid in warding off self-contemplation, brooding and morbid feeling, the nursing of delusional notions, and the ill effects of hallucinatory states. Order, discipline, and regularity of employment do much for these cases; whilst a nutritive unstimulating diet, cold shower baths, and open air exercise are potent adjuvants to treatment. Perhaps in no cases is isolation so vicious as it is here; it is equalled only by association with the chronic insane, which is enforced by our modern methods of overcrowding and defective classification. Mimicry is rampant in these subjects; hence, the sooner they are brought to employ their energies in useful out-door employment which distracts their attention, the better. Amusements, social gatherings, games of chance and skill, and athletic sports should all be encouraged in these subjects, who otherwise spend their existence in isolation from their fellows and in morbid brooding.

Excitement of General Paralysis.—We can only deal with such cases satisfactorily with the complete armamentarium of an asylum at our command. The padded room is often required to obviate self-injury from blind and aimless violence, and to secure them from the injuries which manual interference would so often entail. In advanced cases

—noisy, struggling, degraded—the single room is an absolute necessity, as is also the bed upon the floor to obviate falls. Wherever noisy excitement is not so great a feature, we may resort to observation night and day with benefit. Great precautions must invariably be taken to avoid fractures of the ribs and other bones, which are preternaturally brittle in this disease, and often in a state of extreme osteo-porosis. It is scarcely necessary again to call attention to the danger of bed-sores in these subjects, to the risk of overlooking a distended and paralysed bladder, or to the treatment of destructive habits so prevalent in this disease; the principles already enunciated should be strictly followed out here (see *Destructive Habits*).

Senile Excitement.—Much that has been said as to general paralysis applies to these cases; we have here to deal with frenzied excitement at times, associated with great debility, often with serious cardio-vascular affections, renal degeneration, and all its accompaniments. Only in very mild and early cases can we hope for success from treatment at home; the padded room, the single room, or constant observation, and the frequent attention of the medical attendant are imperatively called for in all serious cases. For the insomnia of senile mania, chloralamide and paraldehyd may be given with benefit; the latter preferably where digestive troubles are prevalent (see *Treatment*, p. 477).

In the later stages of senile decrepitude where excitement has been replaced by mere restlessness, or apathetic dementia, ordinary principles must guide our treatment, and, above all, is it important to give a due amount of rest, and to intervene occasionally where alcoholic stimulation seems desirable.

Alcoholic Excitement.—In the acute non-febrile form—mania-a-potu, or acute alcoholic delirium—the cunning, treacherous, impulsive violence, the delusions of suspicion and persecution, are the features ever to be borne in mind, and they must be treated on the general principles already enunciated (see *Delusions, &c., ante*). The excitement and insomnia are best met by chloral, which is also our sheet-anchor in the treatment of the febrile form of alcoholism or delirium tremens. Duboisine has been given with success, but trional always fails; nor can we trust to cannabis for good results, nor yet to hyoscyamine, which relieves the excitement of chronic alcoholism (see *Treatment*, p. 480).

Therapeutic Measures.—The treatment of insanity by drugs, electricity, hydrotherapeutics, and other measures rests as yet upon an unsatisfactory basis, in so far as it is to a great extent palliative only, and, at the same time, empirical. Our remedies are very largely applied to the treatment of mere symptoms, or we endeavour to establish nutritive equilibrium; or, again, we attempt the removal of such disordered functions in other organs than the brain as may be regarded either as the direct outcome of centric disturbance, or even as the possible exciting cause of the mental derangement itself.

It is too early as yet to base our treatment (as many have done) upon an assumed connection of mental derangements with various visceral disturbances; and though the latter afford indications for treatment, we are far from recognising this principle as the basis for a scientific treatment of the insanities. As we learn more of the agencies which affect tissue metabolism; when we can follow more clearly the products of such activity—constructive or destructive—or, when we awake from our ignorance of the toxines which pathogenic organisms may develop in the system, we shall be nearing a time of rational therapeutics. Physiological chemistry must therefore lead the way towards this wished-for consummation.

We shall not attempt here to do more than merely indicate the *rôle* of the more potent remedies used in the treatment of insanity, the greater number of which are, of course, sedatives and hypnotics. An attempt to treat of the applications of electricity, baths, massage, &c., would unduly extend the space at our command, and we, therefore, proceed to enumerate the chief remedies employed for the treatment of insomnia, excitement, and depression arising in the course of mental disease.

Chloral Hydrate.—The *rôle* of chloral in insanity is chiefly limited to the treatment of sleeplessness in acute insanity, delirium tremens, general paralysis, and the furor and convulsive seizures of epileptic insanity. Its continued use in acute maniacal attacks is to be deprecated as highly prejudicial to ultimate recovery, but in acute melancholic cases it has proved most serviceable.

As a hypnotic we, of course, possess in it a most valued agent, and in all convulsive cases of excitement, such as alcoholic, epileptic, and the excitement of general paralysis, we find it invaluable. In senile cases, however, its *rôle* is far more limited, especially with well-marked arterial degeneration and cardiac debility. In these latter instances it should never be given unshielded by a sympathetic stimulant, such as atropine or alcohol. Given in these cases with stimulants, however, it is apt to produce severe headache next day, although the powerful odour of the breath indicates that rapid elimination is induced. In all cases where we recognise cardiac debility, diffusible stimulants should be combined; but it is wise to discard its use for other hypnotics where there is arterial degeneration, valvular disease, and a tendency to renal degeneration. Undoubtedly, it is of value in melancholia, not for continuous use but for the insomnia, especially if the latter results from aural hallucinations, and where these prompt to desperate suicidal attempts. In the agitated form of melancholia we have seen much benefit accrue from its use when combined with bromides and administered thus two or three times a day, of course, in moderate dose. Here refusal of food will often present itself, and forcible alimentation may be required in addition. On the other hand, it is useless in delusional forms of melancholia where the delusion prompts to the forced abstinence; and in acute mania, except that dependent upon general paralysis, alcoholic insanity, and epilepsy, we should reject chloral, except as a hypnotic for occasional use.

We have already alluded to its use in the gentle excitement of *G. P.*, with evidence of much cerebral excitation and grinding of teeth; here very small doses are most valuable. The rubbing off of the hair, the boring of the head into the pillow, the heated scalp and flushed face, and the tossing of the head to and fro, often accompanied by a sharp cry, all seem to us to indicate its use. In like manner it may be given in the restlessness of choreic insanity with undoubted benefit.

Its use as a hypnotic is also fully justified in chronic alcoholism where delusions of persecution and acutely painful auditory hallucinations render sleep impossible. In delirium tremens it is certainly our sheet anchor. It is far preferable to opium, and should be given in a full dose so as to obtain a long and refreshing sleep. Over and over again have we seen the best results ensue from this treatment; the patient awaking from his slumber a new creature, rational, confident, free from hallucinatory states, and practically in his right mind. In fact, feeding and chloral are the chief agencies to be dependent upon. The great loss of heat after chloral administration should always be borne in mind; unless care be enjoined to prevent exposure, pneumonia is very apt to supervene in debilitated subjects; and it should specially be enjoined upon the nurse that all patients taking chloral at night must receive extra attention in the way of clothing when out-door exercising during the ensuing twenty-four hours.

The extreme value of this drug in epilepsy during the furor, or for averting convulsions, or for the treatment of the epileptic status has already received attention at our hands (see *Epileptic Insanity*), and it remains only to observe that chloral will check the convulsions, wholly or in part, according to the dose administered; a small dose will simply modify the discharge in its spread and intensity, where a stronger dose would entirely arrest the convulsion. A prolonged *heat discharge* has also been observed by us as the result of the convulsive seizure being arrested—a sort of substitution of thermal for motor phenomena.

Chloralamide.—This is undoubtedly a most valuable hypnotic, free from the unpleasant taste of paraldehyd, and from the disagreeable tainting of the breath entailed by the latter. It is fairly prompt in action, in this respect being superior to sulphonal (*Gordon*), but less so than chloral, usually in forty minutes to an hour. Very rarely are unpleasant after-effects experienced, and certainly no digestive or gastro-intestinal derangement; nor is there any lowering of body temperature. Chloralamide does not affect peripheral sensibility; it diminishes reflex action, but does not depress cardiac, respiratory, or cerebral centres, nor is the blood-pressure notably lowered (*Gordon*). As an analgesic it is of little or no service.

This drug is of most service where chloral would be given were it not contra-indicated by cardiac or vascular lesions, where valvular or myocardial degeneration, or where renal disease with more or less vascular atheroma intervenes; here this drug may be given safely, and often with the result of procuring a calm, deep, and refreshing sleep. In the insomnia of melancholia generally, we have found it *most* useful, but its value is also great in the wakefulness of alcoholic insanity and general paralysis. It has by some been also lauded for all forms of acute maniacal excitement. In senile insomnia its value is undoubted. Either sulphonal, trional, or chloralamide may be given in lieu of paraldehyd or chloral, in cases where laryngeal phthisis or inflammatory conditions of the throat or stomach preclude the use of the latter. It may best be given in similar doses to chloral in an alcoholic stimulant in which it readily dissolves, but must never be combined with alkalies or in hot solutions, as it readily decomposes under these conditions.

Paraldehyd. — Prompt in action, even more so than chloral, this drug is a valuable hypnotic, producing in from five to twenty minutes a calm, peaceful sleep, sometimes, however, preceded by slight excitement. Unpleasant effects, such as drowsiness, nausea, headache, rarely occur. It has been shown by Gordon* that even in minute doses it aids the peptone forming power of pepsine, always accelerating the digestion of fibrin, whilst this process is notably *retarded* by chloralamide, sulphonal, and urethane. It, therefore, may be given with more confidence than the latter during the period of food digestion, It has been affirmed that the hæmoglobin is reduced by this drug, methæmoglobin being produced; but this statement has been called in question by Hayem. Like sulphonal and urethane it diminishes peripheral sensibility; slows, but strengthens, the heart's action; and does not depress the respiratory centre. It is, therefore, far better adapted for cases of insanity complicated by organic diseases of the heart and vascular degeneration than chloral, which should be avoided here. It has, moreover, a powerful diuretic effect and has no poisonous action on the tissues.

For the insomnia of senile mania, either it or chloralamide may be given with great hope of success, and with no fear of disagreeable after effects. Its analgesic effect is so trifling that no reliance can be placed upon it unless combined, as it then may be, with morphia. In simple maniacal states it is a most valuable hypnotic, but not so useful as chloral either in the mania attending general paralysis or epilepsy; and, despite the statements of certain authorities, we have found it far less useful in acute alcoholism than chloral hydrate. It can be administered at times advantageously *per rectum*. The great objection to this drug is its pungent taste and the exceedingly disagreeable and persistent odour of the breath, retained often over twenty-four hours, and similarly occasioned when the drug is administered by rectum.

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* *Brit. Med. Journ.*, July 18, 1891.

Disulphones.—Among this group we have at least one most reliable hypnotic. With respect to the individual merits of the three, sulphonal, trional, and tetronal, the latter has failed to realise the theoretical expectation that, having four ethyl groups to the molecule, its hypnotic value would be far greater than the two former; despite the contrary view of Ramoni, clinical experience is generally adverse to this *a priori* dictum. Tetronal, however, is a good sedative (*Schaefer*, *Boettiger*), but is as little adapted as trional as an analgesic, or in cases where the psychical and motor exitement are very severe.

We do not hesitate to regard trional as far the safer and best hypnotic of the group for cases of mental derangement; it is more readily decomposed in the system and more rapidly eliminated than sulphonal; hence its action is more rapid (twenty minutes to an hour) and effective, and its effects less prolonged; the sleep is deep, dreamless, and refreshing (six to eight hours or longer); not attended by the early irregular respiratory rhythm seen when sulphonal or chloral have been given, nor does it entail, like these, weariness, lassitude, confusion, and dreamy states. Lastly, cumulative action has not been observed, and only three or four cases are quoted (even these open to doubt) where hæmatoporphyrin has appeared in the urine; nor has any evidence been forthcoming to prove that the drug exerts any ill effects on the bodily organs.

As a hypnotic we certainly have no safer remedy than trional in most cases of sleeplessness from melancholic depression, either with or without hallucinations, in neurasthenia and in epileptic wakefulness. In general paralysis it is less useful than chloral; and in alcoholic delirium it almost invariably fails (*Köster*, *Boettiger*). We must always bear in mind the fact that where neuralgic and painful affections generally prevail trional is not the hypnotic to be given; or, if it be for any special reason chosen, it should be combined with morphia or some other analgesic. Owing to the fact that its beneficial effect seems to extend to the next evening—a *natural* sleep being usually obtained—some authorities (*Krafft-Ebing*, &c.) recommend it to be given on alternate nights, or it may be varied, with benefit, by other hypnotics, sulphonal, paraldehyd, chloral, or chloralamide; we regard this as an important feature in treatment.

As to its more continued use, the most important *rôle* is, we think, that advocated by Collatz * of giving small doses, say, 7 grains in combination with opium in simple melancholia; in this way most valuable sedative results have ensued. Impulsive forms of insanity are less benefited by it than by chloral or hyoscyamine, nor does it act satisfactorily in the excitement of general paralysis. One great feature is that no injurious action results in cases of cardiac debility, or valvular affection with failing compensation; here, where we dare not give chloral with impunity, we administer sulphonal or trional with absolute confidence. Weir Mitchell has extended its *rôle* to epilepsy; it appears to arrest the fits when bromide is for a time contra-indicated, or when temporary suspension during bromism is called for. In like manner it has been found useful in the restlessness of severe chorea associated with insanity.

The maximum dose of the drug should be considered to be 25 grains, but 15 grains is usually effectual; it should be administered in warm milk or other fluid, or in warm spirits; and, should the effect be unusually delayed, it may with advantage be given a short time before retiring to rest. It is also important to note that it is equally effectual when given *per rectum*.

Hyoscyamine.—This powerful alkaloid, which is isomeric with hyoscine and atropine, has been found of far greater service than the last in the treatment of insanity. It was investigated and extensively employed by the late Dr. Robert Lawson, and his essay upon the physiological effects on the lower animals and man still remains the most reliable and classic contribution to the subject.† He used it in the form of Merck's amorphous extract, given either by the mouth or subcutaneously. It, as well as hyoscine, has been employed very extensively since this date, both in England and on the Continent, and our views as to its efficacy are now fairly clear.

In moderate doses there is a primary lowering of the pulse-rate, followed, subsequently, by a greatly increased rapidity which is long maintained; but if very small doses be given, the depressive effect alone is witnessed and no stimulation occurs. If, on the other hand, large doses be administered, immediate stimulation of the heart's action takes place with no primary depression (*Lawson*). The physiological results of a moderate dose are—well-marked mydriasis in from three to five minutes, reaching its maximum in twenty minutes, and slowly subsiding, it may be, for several successive days. The rise of the pulse-rate is associated with a lowered respiratory rhythm, and a fall in body temperature. The mouth and fauces become dry at an early stage, the secretions generally, with the exception of the renal, are diminished; as regards the latter, there is more or less diuresis. Giddiness and drowsiness are apt to prevail, and sleep is prone to be restless and disturbed. Mild excitement supervenes, attended with rather vivid hallucinations of sight and hearing, and great motor impairment affects the limbs. Speech is somewhat thick and characterised by rambling incoherence.

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* *Berl. Klin. Woch.*, Oct., 1893.

† *West Riding Asylum Medical Reports*, vol. v., 1875.

Hyoscyamine has proved beneficial in reducing the excitement of simple and recurrent forms of mania, and also the maniacal excitement associated with epilepsy. It has been stated to have acted well in reducing the number of fits in the epileptic status; but in view of the undoubted efficacy of chloral here, it is scarcely admissible for this purpose, and our experience would be distinctly adverse to its employment. In chronic alcoholism it has been found useful, and in delusional insanity not of an acute type it has been much lauded (*Lawson*). Certain *symptoms* of insanity are well treated by this alkaloid, and the greatest benefit has accrued from its administration in cases of incessant garrulity or logorrhœa, in the restlessness of senile mania, and in destructive tendencies, whether voluntary or incontrollable. The disadvantage of the drug, which should always be borne in mind, is the tendency to cause much dryness of mouth and throat, and to impair appetite when given in single large doses; hence it is unsuitable where forcible feeding is necessary, or where loss of appetite and gastric disturbance prevail. It is, however, claimed for this alkaloid that *continued* use is not attended by these unsatisfactory results. Again, it should not be given where vascular disease is suspected, especially where extensive atheroma exists. Two cases of hæmatemesis appeared to be very clearly traceable to its administration under these conditions. It must not be forgotten that its long-continued use involves serious loss of body-weight.

So also in melancholia, it is decidedly objectionable, and has always failed here in our hands. Whilst, therefore, its use is mostly restricted to maniacal conditions, it is in the milder and sub-acute forms of mania that it is of chief value; in very acute excitement it is contra-indicated and *a fortiori* in the form of acute delirious mania. As a hypnotic its action is far more powerful than atropine, but the alkaloid hyoscine, which is stated to have five times the power of hyoscyamine (*Martindale*),[*] is more suitable for this purpose in doses of from $\frac{1}{150}$ to $\frac{1}{80}$ of a grain. The advantage claimed for hyoscine is the freedom from after effects, and especially the absence of constipation incurred (*Ringer* and *Sainsbury*).[†] Hyoscyamine is of great value for its moral effect as an agent in the treatment of the criminal type of insanity; this was long since maintained by Lawson, and our further experience of the drug certainly emphasises this opinion. Whether this result ensues from the moral effect wholly is open to question; certain it is that most impulsive forms of insanity of this class are benefited by hyoscyamine, and the consciousness of the motor impotence induced may well play the chief factor towards the favourable issue.

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[*] *Brit. Med. Journ.*, vol. ii., 1888, p. 736.

[†] *Dictionary of Psychological Medicine*, Hack Tuke, vol. ii., p. 1142.

Savage regards Merck's extractive as a useful form of restraint in violent and dangerous cases, "especially those that are very homicidal, and those that, seeming to have lost common sensibility, dash themselves about and run serious risk of injuring themselves." *

Duboisine, said to be identical with hyoscine, has been brought forward as a powerful sedative in conditions of maniacal excitement. Some authorities give the sulphate by injection in doses as large as 1 to 2 milligrammes twice daily; but it is more prudent to limit the dose to 1 milligramme = ·001 gramme, to commence at 2 decimilligrammes = ·0002, and rarely exceed ·0008 gramme. When large doses, such as ·001 to ·002 gramme, have been given, loss of appetite, vomiting, and emaciation have ensued (*Mongeri*, &c.).

After subcutaneous injection of duboisine, mydriasis rapidly ensues, the pulse rate is lowered, motor enfeeblement occurs, and a profound sleep of several hours usually follows in suitable cases.

It has been given as a sedative in acute mania, chronic mania, acute alcoholism, and general paralysis with favourable results; but Mendel asserts that the drug acts by removing all restlessness, and not as a direct soporific, and hence, whilst it is useless in melancholia, paranoia, &c., it is very valuable in excitement with much motor restlessness, unless the latter be due to hallucinations or delusions. † Mendel administers it in doses of from 5 to 8 decimilligrammes. Duboisine has also been given with the best results in hystero-epilepsy by Albertoni and Belmondo, and also in paralysis agitans.

Our own experience is not in favour of its use as a substitute for hyoscine; the latter, or the hyoscyamine extract of Merck, is, in our opinion, of greater value and more extended application.

Opium.—Our experience of the treatment of insanity by opium and its several alkaloids demands from us the expression of unqualified approval. Granting that the cases be carefully selected, we know no drug in the pharmacopœia which is more directly valuable for the cure of certain forms of depression. A fairly large experience has now indicated to us that opium—and opium alone—has any powerfully curative influence over such melancholic reductions as are attended by notable restlessness, mental disquiet, anxious fears, and depressing delusions. As a soporific, except where pain intervenes, we rarely or never employ opium; we have far more valued remedies at our disposal. Its *rôle*, however, apart from its analgesic properties, is that of continuous administration in slowly increasing doses for the relief of painful mental depression, though not for the depression characterised solely by anergic and stuporose states. In these cases, then, of simple melancholia, with much motor restlessness, opium or morphia will not only relieve the depression, but will also often result in no disturbance of stomach or bowel; the appetite will often improve, and the bowels remain free from constipation. The opinion that if given for any length of time it invariably does harm by its effects on assimilation, as stated by Ringer and Sainsbury,* is directly contradicted by the results of our experience in the treatment of depression, which, at all events, should be excepted from the verdict. Owing to its effects on the urinary secretion, many withhold its administration when albuminuria exists, and would strictly prohibit its use in confirmed renal disease. We, however, fully subscribe to the opinion of the last-named writers, that the dangers accruing from its use in these morbid conditions have been much overstated.

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* "Hyoscyamine and its Uses," *Journ. Mental Sc.*, July, 1879, p. 183.

† *Neur. Central.*, February, 1893.

On the other hand, in cases of maniacal excitement we find opium nearly always inadmissible; it usually adds to the excitement, impairs the appetite, deranges peristalsis; why its employment here is followed by such totally different results we know not, but, as a matter of clinical experience, such certainly appears to us an established fact.

In former days opium was given far more frequently as a soporific, but even then it was taught that where excitement prevailed and evidence of "cerebral congestion" was forthcoming it was contraindicated. At the present time we should wholly discard opium for this purpose and fall back upon one or other of the more recent and reliable hypnotics. We prefer giving the drug in the form of the liquor opii sedativus, which is best tolerated by its admixture with an equal amount of sulphuric ether, commencing with doses of 10 to 15 minims twice daily, increasing the dose to 20, 25, and 30 minims three times daily, and rarely, if ever, exceeding 40 minims for a dose. Bearing in mind the idiosyncrasies of advanced age we may safely administer, in this manner, a maximum of 30 minims three times a day, or an equivalent of 12 grains of solid opium. The tolerance established for this drug is remarkable in states of mental depression, and when, eventually, improvement in the symptoms supervenes, it is strange to note how little tendency to habit is engendered and how well the subject stands the immediate withdrawal of the customary sedative. In fact, we can by no means reason from its operation in the sane to its effects upon the insane mind. Should it tend to impairment of appetite, furred tongue, nausea, or constipation we may with advantage substitute morphia subcutaneously, but we shall obtain less of the stimulant effect of opium by this course. The hypodermic administration of morphia is useful when the patient refuses medicine by the mouth, and its prompt operation is one great advantage in its favour. We should not, of course, think of giving opium when there is marked derangement of the digestive organs means should be adopted for restoring the tone and functional efficiency of the digestive tract before commencing a course of opiates; the hepatic functions especially call for attention in this connection, and it may be necessary to wash out the stomach, to administer a mercurial purge, to give naphthaline, beta-naphthol, salicylate of bismuth, or other intestinal disinfectant ere more special treatment be adopted. It may be laid down as a rule that in all cases where forcible feeding is required opium treatment should be postponed.

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* Tuke's *Dict. Psychol. Med.*, vol. ii., Art. "Sedatives."

There are two affections where we should never attempt opium or its alkaloids; we refer to acute delirious mania and to general paralysis. In the former, the adynamic symptoms are almost certain to be increased to an alarming extent if opiates be given; nor is even a single dose as a hypnotic to be given here so long as we have such reliable agents as chloral and the disulphones. In both the climacteric and senile forms of melancholia, Savage speaks favourably of morphia in conjunction with other agencies.*

Cannabis Indica.—Indian hemp has had a very extensive trial in the treatment of insanity, and is undoubtedly a valuable sedative, free from many of the objections attached to other remedies of this class. In full doses the drug causes great exhilaration and a condition of reverie with a train of mental and nervous phenomena of a most pleasurable nature; vivid hallucinations prevail, and the gay and pleasurable state may approach to a stage of ecstasy. Wood gives a lively description of his personal experiences after taking the drug. The above noted symptoms were followed by numbness, by "spells" of partial unconsciousness to his surroundings, time was prolonged indefinitely—seconds seemed hours, distance was exaggerated, and a horrible sense of impending death supervened. Antagonism between his feelings and will was noted, passing into unutterable despair.† Many of these phenomena are experienced during the inhalation of nitrous oxide gas; the prolongation of time, the exaggeration of sounds, the feeling of utter helplessness and despair will be familiar to many who have experienced the effects of this gas. The antagonism referred to by Wood is in this case referred to an outside factor inimical to the personality rather than of the nature of dual consciousness. The drug has a powerful diuretic effect, and this has been suggested as playing an important part in the avoidance of bromism when cannabis and bromide salts are given in combination. The breathing and circulation are but slightly affected, and, although the symptoms of intoxication are so alarming to the patient, the administration of the drug can scarcely be regarded as attended by any real danger (*Wood*).

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* *Insanity and Allied Neuroses*, by Geo. H. Savage, p. 200.

† *Treatise on Therapeutics*, Art. "Cannabis."

A great disadvantage attached to this remedy, as also to conium, is the great variation in strength of different specimens, and the consequent uncertainty of their action; some specimens are quite inert. Dr. Blandford found the extract vary immensely in efficiency.*

With respect to the therapeutics of this drug we have found it of extreme value both as sedative and hypnotic in cases of maniacal excitement; but we have always administered it for this purpose in combination with bromide of potassium (30 grains of the latter to 1 drachm of the tincture). Its *rôle* seems chiefly to be that of reducing the excitement of chronic mania, more especially such cases as present hostile, vindictive, and homicidal violence; the drug appears in every sense a deterrent, without inducing unpleasant and painful feelings, and where the latter exist as the outcome of persecutory delusions a calmative influence is distinctly manifest. We are of opinion that the antagonism occurring in acute intoxication by the drug is mildly represented in the continuous administration of the smaller doses in insanity, and that a feeling of timidity and distrust of their own powers is often engendered and leads to a healthier and more rational adaptation in conduct. The class of patients to which we now refer derive benefit from a long course of the "green mixture" as we term the combination of bromide and hemp; the appetite improves, the body-weight is maintained, even if there be early loss, muscular tone is not impaired, at the same time excitement abates, sleep is more uniform and complete, and the bodily functions are not disturbed. In other forms of excitement, such as the restlessness of senile insomnia, it is also advocated, and even for the sleeplessness of general paralysis (*Dr. Russell Reynolds*). We cannot say that we have confirmed this latter statement, but are quite convinced that its use, as Dr. Reynolds states, in cases of acute mania and alcoholic delirium is at least restricted, if not absolutely contra-indicated. On one point most authorities appear agreed; it is injurious in cases of melancholic depression, especially if given in continued doses.

In epileptic mania, cannabis in combination with bromide is of the greatest value, and, in fact, in the whole list of the explosive neuroses this combination is most suitable. It also finds its *rôle* in the excitement and vicious turbulence of imbecility; still more so when the subject is an epileptic. It will be seen from what preceded that, like hyoscyamine, we find cannabis most serviceable in chronic and incurable forms of insanity, and that to a certain extent it is interchangeable with the former in the treatment of these forms of disease.

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* *Insanity and its Treatment*, 4th edit., p. 440.

It is well known that cannabis in the form of hashisch is the cause assigned for a large proportion of the insanity of the asylums in India and elsewhere; thus at the Cairo Lunatic Asylum Dr. Sandwith found that out of a population of 226 males and 62 females, there were as many as 23 recent cases under treatment for hashisch, and 150 other chronic dements whose insanity was traced to the same cause.*

It is of interest also to note that Dr. Sandwith alludes to the ever *increasing timidity of* those accustomed to its continuous use, and which may partly explain the moral influence which this drug certainly exerts in subjects of chronic mania.

Conium.—This drug was formerly used far more extensively than it is at present, and its disuse probably is accounted for on the same grounds which led to its discontinuance by ourselves, viz., its extraordinary variability in physiological operation, and the absolute uncertainty of numerous samples of the drug vended. Frequently the preparations were quite inert, no trace of the usual physiological effects being presented when large doses were administered.

This uncertainty of operation has proved fatal to its retention as a depresso-motor for cases of insanity, in view of the far readier and more certain effect of other agents which may now be employed. Its physiological action is slowly established even under large doses; the pupils dilate, vision may be disturbed from paralysis of accommodation, some giddiness may be experienced, but the most marked symptom is great languor and muscular debility, arising from paralysis not of the trunks of the motor nerves so much as of the motor end-plates. The influence on the circulation is trivial, the pulse, at first depressed, becomes somewhat quickened. The sensory nerves are not affected by medicinal doses; if lethal doses are given, tingling, numbness, and formication have been complained of.

We have found this drug of value in simple mania, and in no other form of mental disturbance. When a trustworthy sample has been secured it certainly diminished the restless activity of the subject, had a powerful calmative influence over the excitement, and fostered the tendency to sleep. We have always administered this drug in the form of succus conii, and in doses of two to six drachms two or three times a day. The alkaloid has also been given subcutaneously and apparently with much success by some. We have, personally, no experience of its administration in this form.

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* *Journ. Mental Sc.*, vol. xxxiv., p. 482.

PART III.—PATHOLOGICAL SECTION.

General Contents.—Morbid Condition of Cranial Bones—Investing Membranes—Brain Substance—Histological Elements of Cortex—Forms of Tissue Degradation—Pathological Anatomy of General Paralysis, of Epilepsy, and of Chronic Alcoholism.

GENERAL PATHOLOGY AND MORBID ANATOMY.

Contents.—The Cranium—Dura Mater—Pia-Arachnoid—Arachnoid Hæmorrhage—Adherent Pia—Vascular Apparatus—Congestion—Inflammation—Softening—Atrophy—Miliary Sclerosis—Colloid Degeneration—Granular Disintegration of Nerve-cells—Pigmentary or Fuscous Degeneration—Developmental Arrest of Nerve-cells—Vacuolation of Cell-protoplasm—Vacuolation of Nucleus—Destruction of Intra-cortical Nerve-fibre Plexus—Tissue Degradation from Over-strain—Tissue Degradation from Active Morbid Processes—Tissue Degradation from Disuse—General Summary.

The Cranium.—The bones of the skull-cap present as their more frequent anomalies of texture, one or other of the following conditions:—(1) They may be thickened even to an excessive degree, and yet be light in weight from the abundance of rarefied diploe. (2) They may be increased in thickness, and heavy from general increase in density throughout, and subperiosteal addition of bone. (3) They may be extremely dense, but not thickened (on the contrary, they may be thinner), and the surfaces eburnated and polished in aspect. (4) They may be reduced in thickness and density, even to such a degree as to become semi-diaphanous over certain regions.

The first condition (due to subjacent irritation) is sometimes associated with thickened and adherent dura mater; **subacute inflammatory** states, probably, explain this association of central rarefaction with superficial hyperostosis. The second condition is far more frequent, and sometimes leads to extremely heavy skull-caps; it may also be due to prolonged and very chronic inflammation of the texture; although in many cases it is probably the result of repeated vascular engorgements, and the excess of nutritive plasma brought to these parts by conditions of violent cerebral excitement, occurring through a period of many years in chronic mania. The thickened dense skull-cap is frequent in **epileptic** subjects, and in the dementia of **chronic insanity.*** The inner surface may exhibit protuberant bosses, frequently coinciding with subjacent atrophy of brain-substance; whilst the grooving of the vitreous table is converted into a deep channelling, extending almost to the cancellated structure, and bridged over here and there by newly-formed bony tissue. The hyperostosis is generally disposed over the whole of the vertex, but is almost invariably most pronounced in the frontal and the occipital regions, and more especially the former locality. When, as we occasionally find, localised hypertrophy occurs, the frontal bone is by much the more frequent site of the thickening. We have records of fifty-four cases of localised hyperostosis, and in thirty-one of these the frontal was the site of this bony increase; in seven cases the occipital, and in six cases the parietal were the regions involved.

A not infrequent disposition, and one which carries with it considerable interest, is the thickened state of the left frontal associated with that of the right occipital regions. The frequency of cranial hyperostosis may be gleaned from the fact, that it presents itself in one-fourth up to one-third of all cases of insanity. Thus, of 1,565 fatal cases of insanity, the cranial bones were thickened in 404 instances (or 25·8 per cent.); and they were indurated, dense, and heavy in 523 subjects, or 33·4 per cent.

The diminution in the thickness and density of the cranial vault is most usually seen in **senile atrophy**; the process whereby such a condition is induced being, in fact, similar to that universally prevailing at this period. The **facial bones,** however, are more subject to this atrophic change than those of the **cranial cavity,** and, in fact, we may note the co-existence of the former with hypertrophic thickening, and induration of the latter (*Rokitansky*). The sutural lines are usually the site of most advanced atrophy, and irregular depressions or pits alongside the sagittal suture indicate the absorption due to exuberant Pacchionian bodies along this course. The vitreous table is more especially involved in these senile cases, and the morbid process is one of eccentric atrophy, the compact being gradually replaced by cancellous tissue. Osteophytes in the form of irregular superficial masses on the inner surface of the cranium, osseous spiculæ, plates, and small exostoses are occasionally met with; as also a form of eburnated osteoma of concentric lamellæ with radiating canaliculi, and devoid of blood-vessels (*Virchow, Cornil and Ranvier*). All these indicate "an extinct localised inflammatory process, the products of which here remain in an ossified form" (*Griesinger*). Our own observations tend (with certain qualifications) to confirm the opinion long since expressed by Dr. Bucknill, that the increased thickness of the cranium in insanity is not connected with cerebral atrophy, his statement being—"Some of the heaviest and thickest crania which we have met with have occurred in instances in which there was little or no cerebral atrophy; and the condition of the cranium where there is undoubted atrophy of the brain, is not unfrequently one of abnormal tenuity."*

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* "Hyperostosis almost always presents itself in both its forms, namely, that of deposition externally upon the bone, and simultaneous condensation of its tissue (sclerosis). In a few cases it goes on to such an extent that the skull is not only (according to Iadelod and Ilg) larger than natural, misshapen, and uncommonly thick (9 lines to 1½ or 2 inches), but it also acquires a weight that is almost incredible."—*Pathol. Anat.*, Rokitansky, vol. iii.

Exostoses and bony spiculæ are exceptionally rare in those dying insane. Dr. Bucknill states that such outgrowths were found by him in but three cases out of 400 subjects. Our own observations would tend to render them of even less frequent occurrence; since out of a total of 2616 fatal cases of insanity exostoses occurred in but six cases, and bony plates in the membranes in eight other cases.

Dura Mater.—This tough inelastic sac investing the brain has so long been considered and described as of double constitution (that is, of dura mater proper, and parietal layer of arachnoid) that it becomes important to define our position respecting its nature ere we describe its anomalies. Rokitansky especially insists upon the distinction—"We are compelled to adopt the distinction by the substantial difference which is exhibited, at least at first, by morbid processes in the two layers. Inflammation, for instance, attacks one of the layers independently of the other, and presents differences accordingly in its course, in its proneness to extend along the surface, and in the products it furnishes, which manifest the analogy between that layer and serous membrane in general."† Despite the assertion of so eminent an authority, the tendency of modern anatomists has been towards the contrary opinion. The epithelial layer, forming the inner smooth surface of the dura mater, is now generally regarded as proper to that membrane, and not, in the true sense of the word, a reflection of arachnoid. One fact, which tended to emphasise the divergence of opinion respecting this anatomical structure, was the frequency of the formation of so-called **arachnoid cysts** within the cavity embraced between dura and visceral arachnoid. A certain number of pathologists regarded their origin as **hæmorrhagic,** pure and simple; another class viewed them as products of true **inflammatory** conditions; and the latter naturally held that the inner surface of dura, to which they are often attached, is a true serous surface, giving rise to these inflammatory exudates. With Axel-Key, and Retzius, we would describe a visceral arachnoid only, so that the structures and spaces formed between the cranial bones and the brain would be from without inwards—(*a*) *dura mater;* (*b*) *sub-dural space* (*formerly the "arachnoid sac"*); (*c*) *arachnoid;* (*d*) *sub-arachnoid cavity;* (*e*) *pia mater;* (*f*) *epicerebral space.*

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* *Psychological Medicine*, Bucknill and Tuke, 3rd edit., p. 566.

† "Pathological Anatomy," *Syd. Soc. Trans.*, vol. iii., p. 323.

It must be remembered that this tough fibrous membrane is firmly attached to the inner surface of the cranium, as its inner periosteum; but, the attachment amounts to firm adhesion along the sutural lines, and at the basal openings, foramen ovale, foramen lacerum, and foramen magnum. It is supplied with blood from the various meningeal branches, and a rich supply of nerves from the fifth, twelfth, and sympathetic; in inflammatory conditions of the bone and of this membrane, the inelastic nature of the dura sets up very acute pain from compression of these nervous filaments (*Duret*).

Adhesion, to a morbid degree, betwixt this membranes and the bones of the cranium is of frequent occurrence in chronic insanity; partial adhesions, indicative of bygone inflammatory change, are found in some 15 per cent. of those dying insane, whilst universal strong adhesions were established in 90 out of 1,565 fatal cases of insanity (a percentage of 5·7). The favourite site, as before stated, for partial adhesions is the frontal bone, either along the course of the coronal suture, or in the hollow corresponding to the external prominences of the frontal bosses (10 per cent.); the next more frequent site is the sagittal line and the parietal bone on either side. In a small proportion of cases, sixteen only (1 per cent.), the chronic inflammatory change had induced a noticeable thickening of the dura; and in a still smaller category was its appearance as to colour modified from the engorgement of its small vessels (in such cases, the texture was softened and infiltrated, and the subjacent bone similarly involved). Morbid adhesion betwixt this membrane and the arachnoid and brain is of very rare occurrence; we have seen it in but ·6 per cent. of our cases; and this we regard as another indication of the nature of the epithelial lining of the dura, which seems to present no morbid sympathy with the true arachnoid or to be liable to adhesive inflammatory states. Rokitansky has affirmed the frequency of such connections, but we fail to verify his statement; at all events, amongst the insane community.* The adhesions are often so extensive and firm that it is difficult to remove the brain without injuring the organ; hence, the skull-cap with its attached dura have to be removed together. On attempting to separate it from the bones in these extreme forms, we fail to do so, as the membrane tears into shreds or splits up into layers, leaving irregular white glistening membranous lamellæ, strongly contrasted with the rosy or deeper-tinted bone around. All this is, of course, indicative of the results of old-standing inflammation.

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* See also on this point Dr. Bucknill: *Psychological Medicine*, Bucknill and Tuke, p. 568.

Bony plates within this membrane, as a result of further change in the inflammatory exudate, occurred in 8 cases out of a total of 2616. It is probable that the exostoses described on the inner surface of the bones of the skull have their origin often in the membrane itself, or in exudate intervening betwixt the two. In one remarkable case the whole of the falx cerebri was thus ossified into a corresponding sickle-shaped bony plate,* such as permanently obtains in the ornithorhynchus; on the other hand, a bony tentorium cerebelli, which we know is normal in many mammals, we have never seen amongst the insane.

The Pia Arachnoid.—A milky cloudiness of the arachnoid is seen in most brains of those dying at middle age, and the opacity becomes more decided with advancing years, until, in the aged, it is seen to a notable degree, apart from any actual cerebral disease. It has been invariably attributed by pathologists to frequent congestive conditions (chronic hyperæmia) of the membrane, to which (as Rokitansky affirms) every one at an advanced age must have been occasionally subject.† Whatever be the explanation in the comparatively *healthy* brain, there is no doubt that in the extreme degrees of this change, seen in the insane, we must infer a chronic inflammatory agency. In senile atrophy of the brain we see this physiological retrogression emphasised, opacities and thickening of texture being often marked features here, apart from any inflammatory change; the outbursts of senile mania are, however, often associated with a chronic meningo-cerebritis, which subsequently reveals itself in morbid adhesions betwixt the brain and soft membranes.

It is peculiarly frequent in those prone to alcoholic intoxication (*Rokitansky, Griesinger*), and we have seen it as a constant change in the brain of the sane and insane criminal class—which class is, to a notorious extent, addicted to intemperance.‡ In the insane community generally, we have found arachnoid opacity prevails to a notable degree—in nearly 50 per cent. (772 out of 1,565 cases)—and, as usual, most marked along the sulci and the immediate vicinity of the blood-vessels.

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* "A similar structure, constituting an unique specimen of anatomical variety, is exhibited in the skull of a female belonging to my collection" (*Blumenbach*).

† For the histological changes of the pia-arachnoid evoking the milky opacity seen in insanity, *cf.* article by Dr. Robertson, *Journ. Mental Sc.*, Oct., 1895.

‡ "It may be generally considered as the result of former chronic hyperæmia and inflammatory stasis; it accordingly occurs together with increase of the Pacchionian granulations—which depends on analogous processes—under all circumstances where habitual cerebral congestion existed during life, as in the case of drunkards, who, indeed, can rarely be considered as mentally healthy." Griesinger on "Mental Diseases," *Syd. Soc.*, 1867, p. 418.

It is usually associated with considerable thickening of the pia arachnoid (the pia is abnormally thickened in fully 48 per cent. dying insane), partly from fibrinous exudates which have organised, partly from plastic lymph, and often from an œdematous swollen condition of the conjoined soft membranes, and the trabeculæ intervening betwixt them in the sulci, resulting in a watery, semi-translucent, gelatinous appearance, with here and there scattered patches, points, or streaks of opacity. The outer surface of the arachnoid, covered with a delicate pavement-epithelium, becomes at times perceptibly rough and granular, both to the touch and to naked-eye examination; the condition resembling, histologically, the granular condition of the ependyma, or lining-membrane of the ventricles; it is especially prone to occur at both sites in general paralysis of the insane.*

The conditions above referred to of opacity with thickening of the pia arachnoid may be unattended with any excess of fluid in the sub-arachnoid space; usually, however, in the chronic insane, great excess prevails, so that the normal limpid cerebro-spinal fluid (which varies considerably between 2 drachms and 2 ounces) may be very largely augmented up to 8 or 10 ounces; the soft membranes are buoyed-up by such accumulation, and their meshes become thickened, glutinous, and water-logged. The fluid is acid in reaction, whilst the normal cerebro-spinal fluid is alkaline. It must be borne in mind that this sub-arachnoid cavity is continuous with the general ventricular cavities of the hemisphere, with the central canal of the spinal cord, and with the sub-arachnoid space; products of inflammatory activity being thus capable of transmission to distant parts by the movements produced in this fluid during locomotion, respiration, and circulation, all of which are known to affect the cerebrum.

In connection with the arachnoid there is an important morbid state, the frequency of which, in insanity, and especially certain *chronic* forms of insanity, makes it a striking feature in our *post mortem* records; the condition referred to is that of false membranous productions, enclosing various contents from straw-coloured serum to thin bloody serum, or blood partly or completely clotted. These formations have been often referred to by the inapt term of arachnoid cysts. When they occur as gelatinous-looking exudates or pseudo-membranous structures upon the inner surface of the dura, they have been regarded as inflammatory in origin, and have been described by Virchow as **pachymeningitis interna,** in accordance with the view of Calmeil, Boyle, and others. We incline strongly to the view that the inflammatory theory of their origin cannot be supported by a tithe of evidence from asylum experience; there can, we think, be little or no doubt that, in the case of those dying insane at least, we must claim a **hæmorrhagic origin** for such formations.[*] Their frequency may be judged of from the fact that 81 cases are recorded in 1,565 autopsies of the insane, and their special frequency in that form of insanity which is associated with general paralysis, is emphasised by the occurrence of 30 instances in a series of 242 general paralytics (a percentage of 12). The appearances presented by them vary considerably with the stage at which they are found. In early stages we may find a slight rusty-staining over a more or less localised patch of the inner surface of the dura, or, perhaps, covering the whole area of one of the fossæ (and especially the middle fossa at the base of the skull); over this rusty-stained groundwork minute droplets of blood are seen, as if besprinkled by a brush, or as if the blood had oozed from the surface by a sort of sweating process. The rusty-staining can be scraped off as an extremely delicate and structureless pellicle. Or, again, the inner surface of the dura may overspread a somewhat amber-coloured glutinous-looking layer, which can be readily stripped off and appears to be a purely fibrinous formation, enclosing more or less arterial blood. Such structures form translucent pellicles, which, when examined, look like gutta-percha tissue stretched to a delicate tenuity. The extravasation is often extensive, flattening the convolutions, and inducing considerable atrophy—a simple dark clot of blood moulded to the form of the arachnoid cavity occupied by it, thick at its central parts, thinning out towards its margin, and covered with a delicate fibrinous layer above and below; or the fibrinous formation may be thicker, more organised, and may constitute a complete sac enclosing the blood-clot. Rokitansky's classical description holds good for the formations in the insane.

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* See "Degenerative Lesions of the Arterial System," by Cecil F. Beadles, *Journ. Mental Sc.*, Jan., 1895, p. 45.

> "Its adhesion with the dura mater, too, is loose; it partly sticks on, and partly is connected with the membrane by a few small vessels. Both walls of the sac are usually of a brown, rusty colour, and tenacious. They may often be separated into several layers which vary in thickness, but the inner of which are more thin; at the margin of the sac they coalesce and form one lamina, which soon becomes reduced to a thin, brown, rusty-coloured membrane, and spreading out further on the cranial vault, reaches to the base, and at length terminates in a thin, rusty-coloured, gauze-like film . . . within, the sac contains a more or less thick fluid, of a dark and various colour, like chocolate, or plum-sauce, rust, or yeast; in course of time the lymph is gradually removed, the inner surface of the sac becomes smooth and polished, and the contents are changed into a colourless, thin, clear, serous fluid."[†]

We have never observed what the same authority states is of occasional occurrence—viz, the ossification or formation of bony plates or concretions on its outer wall next the dura. Some of the more cogent reasons for regarding these formations as non-inflammatory are:—

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[*] See "The Pathology of Sub-dural Membranes," by Ford Robertson, *Journ. Mental Sc.*, vol. iv., July, 1896.

[†] *Op. cit.*, p. 330.

1. The cyst is readily removable, slightly (or not at all) adherent to the dura mater.

2. In the majority of cases there is no evidence whatever of the existence of a pachymeningitis; (*a*) the dura is not thickened or softened, or vascular; (*b*) no organic connection exists betwixt the two.

3. In early stages the characters are purely those of a simple extravasation of blood into the arachnoid cavity (subdural space).

4. There is the co-existence in this affection of a recognised vascular disease and vasomotor disturbances which render hæmorrhage frequent—*e.g.*, the othæmatoma or "insane ear."

In an important communication my former colleague, Dr. Robert Lawson, expressed the same views as are here entertained.*

As regards their cause and origin and general etiological relationship amongst the insane, it has been pointed out by Sir J. Crichton-Browne that the *age* of their more frequent occurrence was between 35 and 45 years—an important feature as distinctive between it and ordinary forms of cerebral hæmorrhage, which occurs more frequently at a much later period of life. The same authority in an analysis of fifty-nine cases of arachnoid cyst, occurring at the West Riding Asylum, has clearly established the vast preponderance of this accident in cases of **general paralysis**, as seen in the following table:—

FIFTY-NINE CASES OF ARACHNOID CYST.

General paralysis affords	29
Chronic disorganisation of brain	16
Senile atrophy	4
Epilepsy	3
Mania associated with chorea	2
Mania with meningitis	2
Insanity with Bright's disease	2
Chronic mania with phthisis	1
	59

As regards sex, it is recognised as occurring more frequently in men, from very obvious reasons; the most important being the greater frequency of its congener, general paralysis, in the male than in the female.

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* Dr. Lawson's statement is to the following effect:—"Amongst the sane, amongst drunkards, and in cases where injury has induced pachymeningitis externa, this production of arachnoid cysts by the rupture of vessels formed in inflammatory products might readily occur. . . . It is evident that at least the large majority of cases of hæmatoma in the insane, originate in direct rupture of vessels and extravasation into the arachnoid sac." (*Brit. and For. Med. Chir. Review*, 1876.)

Our records embrace the histories of 73 additional cases since the above were tabulated by Crichton-Browne, and these, arranged as to associated cerebral states and sex, illustrate forcibly the foregoing statements:—

SEVENTY-THREE CASES OF ARACHNOID CYST.

General paralysis afforded instances	in	30	Males	and	4	Females.
Chronic disorganisation of the brain	,,	12	,,	,,	7	,,
Senile atrophy . . .	,,	6	,,	,,	2	,,
Epilepsy	,,	3	,,	,,	1	,,
Mania	,,	2	,,	,,	2	,,
Melancholia . . .	,,	1	,,	,,	2	,,
Idiocy	,,	1	,,		...	,,
		55			18	

On summarising both series we have 132 instances of arachnoid cyst; 63, or 47·7 per cent., occur in general paralysis; 35, or 26·5 per cent., in chronic disorganisation of the brain; 12, or 9 per cent., in senile atrophy; 7, or 5·3 per cent., in epilepsy; and 15, or 11·3 per cent., in several other forms of mental ailment.

The site of hæmorrhage is almost exclusively confined to the vertex and lateral aspects of the cerebrum; we have never seen it on the lower aspect of the tentorium or within the cerebellar fossa of the cranium; it is also of rare occurrence in the anterior and middle fossæ at the base.

The *left* hemisphere is more frequently the site of the hæmorrhage than the right, as indicated in the sixty-five more recent cases extracted from our records.

SITE OF ARACHNOID HÆMORRHAGE IN SIXTY-FIVE CASES.

Both hemispheres generally covered above . .	=	28
Left hemisphere ,, ,, ,, . .	=	20
Right hemisphere ,, ,, ,, . .	=	11
Right parieto-occipital region	=	2
Right frontal region	=	1
Anterior fossa at base	=	1
Anterior and middle fossæ	=	2
		65

The extravasation most probably occurs from a vessel of the pia mater, the vessels of which in general paralysis show very special lesions forming one of the most constant changes in this affection. The diseased condition of their tunics, which we shall allude to later on, is greatly aggravated by the repeated, violent, and long-continued outbursts of excitement to which such cases are subject; and the vessels which have undergone most change are decidedly those in the anterior regions of the brain, coinciding with the more frequent site of these arachnoid hæmorrhages. The arteries coursing within the sulci are, of course, better protected by the support they receive, than the veins distributed over the exposed surface of the gyri; at the summits of the gyri there is evidence of inflammatory activity, well marked in the presence of meningeal adhesions. The vascular network supported by the pia at these sites is most affected by the morbid changes taking place, and hence, the venules are kept in a state of continuous engorgement near the site where they empty into the larger veins running to the sinuses, a condition further aggravated by the active arterial flow of functional excitement. Thus, in our opinion, the occurrence of at least a large proportion of these formations is explained as due to :—

(*a*) The initial diseased condition of the vascular tunics.

(*b*) The distended condition of the venous system from atrophy, and consequent loss of support, and obstructive conditions due to inflammatory change.

(*c*) The anatomical arrangement of the veins, involving them in the most pronounced inflammatory change.

We have alluded to the evidence of congestive and chronic inflammatory conditions presented in the notable opacity, with thickening of the soft membranes, the presence of effused lymph, and, we might add, the somewhat rare condition of purulent infiltration of the membranes at the vertex in certain forms of insanity. To these we must add, as indubitable evidence of inflammatory activity in the cortex and its investing membranes (chronic meningo-cerebritis), the frequency of morbid adhesions between these structures. Such morbid adhesions occur in chronic insanity, in chronic mania, in senile mania, occasionally in alcoholic insanity, and especially in the mental derangement associated with traumatism. It is, however, in general paralysis of the insane that this condition forms so important a feature as to constitute the one distinctive sign indicative of this disease to the pathologist. In a small percentage of cases only is this important sign absent; but in such exceptional forms other indications are sufficiently expressive of the nature of the diseased process. We shall describe in detail the morbid process as it occurs when dealing with the special pathology of general paralysis. For the present we have to deal with the general features presented by such adhesions in this and other forms of mental disease. I find from Dr. Bullen's statistical compilation from our West Riding Asylum Records, that out of 1,565 fatal cases of insanity, morbid adhesions betwixt brain-cortex and investing membranes had been contracted in 340 instances, or 21·7 per cent. of the whole; whereas in a former study of general paralysis, some years back, we found that in 241 cases of this disease which had proved fatal, 186 (or over 77·1 per cent.) presented well-marked adhe the remaining fifty-five (or 22·8 per cent.) being described as free such implication. We must, however, be prepared to meet cases where it may be dubious how far we should regard connection as amounting to a morbid adhesion ; but in our stati results above recorded, we include only such instances where removal of the membranes necessitates a *tearing away* of the super cortex. Undoubtedly, many other cases of morbid firmness of v present themselves, which, therefore, escape from this category ; this was the case in many of the 22·8 per cent. in which *ge adhesions* were excluded. The brain of the alcoholic presents inst of this morbid firmness of union ; and, histologically, this is att to in the presence of the same morbid elements which are fou profusely scattered through the cortex in genuine cases of adhe but we do not find in the alcoholic's brain, as a rule, anything than this undue firmness of union.

On attempting to strip off a portion of adherent membrane, are seen by the naked eye numerous tough fibrous prolongations, v look like enlarged blood-vessels, connecting the under-surface o pia with the cortex of the brain. When forcibly removed, the layers peel away to varying depths upon the pia, leaving an e surface which presents a highly characteristic aspect. The su looks gnawed or worm-eaten along the length of the gyri with irre sinuous margins, so that it somewhat resembles the aspect sented by a succulent leaf which has been attacked by a cater The base of the eroded (or rather, torn) surface is distinctly punc by large open orifices from which coarse vessels have been withd Adhesions of some age exhibit a coarse dense fibrillar conne betwixt pia and cortex; the normally delicate retiform aspect c neuroglia is lost in the coarse fibrillation which has ensued. In e stages the appearance is suggestive of inflammatory implication, i distinctly-pinkish appearance of the cortex, sometimes diffused, times limited to the areas of recent adhesions ; the pia is thicken tumid, the seat of nuclear proliferation, its vessels deeply eng and the superjacent arachnoid also thickened, opaque, and œdem The distended vessels are coarse and tortuous, their sheaths thic by multiplication of their cells and the traversing of their structu wandering leucocytes. The microscope reveals infiltration of the c by large numbers of peculiar spider-like cells—oval, flask-shap globose—but all throwing off numerous delicate fibrillar processes entwine upon the vascular walls and meander amongst the elements of the cortex. Such spider-like cells are found in *all* adhesions in the upper layers of the cortex, immediately beneat adherent pia, forming a direct connection with its under s and the vessels passing from it into the substance of the brain. Around the walls of the blood-vessels these elements tend especially to crowd, and their ramifying extensions will, probably, by subsequent contraction, seriously interfere with the *permeability of these channels of nutrient supply.* The prominent *rôle* assumed by these organisms in general paralysis of the insane, the frequency with which they are seen, and their very striking features, induced certain observers to regard them as pathognomonic of this disease. We had, however, some years previously indicated and sketched their appearance in **senile atrophy** of the brain (*Pl.* xxiii., *fig.* 1), and had recognised their existence in other morbid conditions; * in fact, they represent a hypertrophied state of what in our section of the normal histology of the cortex we have described as its "lymph-connective" system.

The reason why these morbid conditions are not more frequently seen in senile atrophy and other pathological states of the brain is that the stage in which they are formed is an *early stage* of the disease, a stage which in most fatal cases has long since passed by; the organisms have succumbed to a fatty liquefaction and so been removed from observation. General paralysis, however, is a comparatively rapid process of dissolution, and intercurrent affections often prove fatal, and afford us illustrations of its morbid anatomy in early stages; hence these morbid appearances are frequently met with, yet not constantly, for, at an advanced stage of this affection also, the morbid cells degenerate and disappear, leaving their fibrous meshworks as their sole representative. In chronic alcoholism, again, such products of morbid activity present themselves, frequently in great abundance, but never, in our experience, to the extreme degrees met with in certain cases of senile atrophy and general paralysis.† In fact, it is our opinion, based upon a large number of observations, that where a specially irritative process is engendered in the cortex, and more especially where a large accumulation of degenerative material has to be carried off from this region, or where effete material accumulates as the result of some obstruction to the normal transit of lymph from the brain, there we are likely to meet with these vast developments of "spider-cells," as they have been termed. Hence in a **chronic meningo-cerebritis,** attended by much effusion into the vascular sheath, by extravasations into the brain-substance, and by the varied products of inflammatory engorgement of the part, this "lymph-connective system" of the neuroglia (as we have ventured to term it) undergoes the functional hypertrophy here alluded to, in an extreme degree.

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* *Proc. Roy. Soc.*, No. 182, 1877.

† See in this connection "The Morbid Changes of the Aged Insane," by Alfred W. Campbell, M.D., *Journ. Mental Sc.*, Oct., 1894, p. 645, in which an attempt is made to distinguish the typical senile spider cell from that of general paralysis and of alcoholic insanity.

Again, in the later stages of senile degeneration, the fatty atrophy of texture has advanced to so extreme a degree, usually during a very prolonged course of many years, that the surface of the cortex is widely severed from the membranes overlying it by the compensatory accumulation of fluid; any delicate adhesions which had been formed in early stages have been softened and broken down. The membranes are not thickened to the same extent as in general paralysis, where they often form a dense, thick, felt-like structure which fully occupies the space formed by the recession of the atrophic brain. The physical conditions, therefore, as well as the more rapid course of the one compared with the other, have probably much to do with the presence or absence of adherent membranes. We must, however, not omit the fact, that the tearing of the cortex is, to a certain extent, also due to the *softening* of the outer layers of the cortex by the *inflammatory process;* but this alone by no means accounts for the appearances, since the condition observed on removing the membranes in a brain simply softened by decomposition, or from those regions always excessively soft, at the basal aspect of the cerebrum, in no way reproduces the appearance of the eroded cortex in general paralysis.

The **brain-substance,** both grey and white, in fatal cases of insanity is found in a very variable condition of vascularity dependent frequently upon wholly-extrinsic agencies and accidental states, which are completely foreign to the cerebral disturbance existing during life. We must remember the peculiarities of the vascular mechanism we are dealing with, which explain to a great extent the variations noticed. The pia mater is a wondrously vascular meshwork, capable of an enormous degree of distension and venous engorgement, as we sometimes see to an astonishing extent in obstructions to the return of venous blood to the heart, in cases of intra-thoracic pressure. It serves the purpose of bringing into immediate contact with the surface of the brain a very large amount of venous blood; the carriers of which are so disposed as to offer a direct mechanical disadvantage to the return of venous blood from the cranial cavity—the current of blood in the large cerebral veins being opposed to that of the current in the sinuses by their oblique direction and opening into the sinuses from behind forwards. Thus, whilst in the veins of the lower extremities, special facilities (such as their valves) are introduced to favour the circulation in its return, the intracranial veins have a direct obstruction offered to the too speedy flow towards the heart; an obstruction which even leads to a hypertrophic state of the tissues in this immediate neighbourhood.* The venous blood in this vascular membrane and system of sinuses serves the purpose of keeping up a sustained backward pressure upon the cortical venules, and thus effectually provides for the continued patency of the minute vessels of the cortex. It is only exceptionally that this patency is interfered with to a state of complete anæmia, when, of course, unconsciousness supervenes. Sleep is an instance of a rhythmic interference with this condition; and the agency whereby the anæmia is produced is well illustrated in Mosso's experiments with the plethysmograph, whereby he clearly shows a well-marked dilatation of the peripheral vessels as the immediate prelude to sleep. A similar condition of things is found at the other extremity of the cerebral circulation—viz., the basilar artery. Here we find the two vertebrals taken together about double the capacity of the recipient basilar artery; and as the result of this a sustained pressure of no inconsiderable degree is kept up in the minute nutrient arteries passing direct from it into the substance of the pons. Hence these vitally-important centres are kept continuously supplied with blood, a supply which will only be augmented as contraction in the distal cerebral branches produces anæmia in those parts. If we keep this mechanism of the venous system of the cortex in view, we at once see how variable will be the vascular appearances of the brain according to the mode of death; the presence of obstruction in the heart and lungs to the venous circulation, and especially obstruction in the cranial sinuses, such as frequently occurs in cases of insanity. So likewise the mode of opening the body for *post-mortem* examination greatly modifies the appearance of the cortex and white matter, but especially of the soft membranes. If the cranium be opened before the thorax, the vessels will be found far more engorged than when the reverse procedure is adopted, whereby opportunity is afforded for draining off the blood from the head through the large vessels so severed in the chest. As to the results of intra-thoracic pressure, we must be prepared to find engorgement of the cerebral vessels in the pia in all cases of severe obstruction or obliteration of the pulmonary blood-vessels—*e.g.*, extensive new growths in the mediastinum, copious pleuritic effusions, constriction from various causes of the roots of the lungs, fibroid induration of lung, &c. Rokitansky has alluded to the extremes we occasionally meet with of this obstructive engorgement of the cerebral membranes, the vessels of which he describes as forming "spirally-twisted coils and intestine-like circonvolutions." * Nor is this any exaggeration of what we see occasionally in asylum practice. We should have said *very rarely*, for but *three* such cases have occurred in our experience of considerably over *two thousand* inspections. Such, for instance, was the case of an alcoholic subject suffering from fibroid induration of the lung, and in whose case capillary bronchitis supervened, resulting in an extremely-stuporose condition for days together. The necropsy revealed an extraordinary development of varices and contorted vessels in the membranes, actually concealing from view extensive areas of the brain-surface; whilst enormous numbers of extravasations varying from miliary and punctiform hæmorrhages to patches from a pea to a florin in extent were scattered throughout a deeply-congested brain both in grey and white substance. † To a much less marked degree is the engorgement recognised in obstructive thrombosis of the sinuses, for in all examples met with we have found the patency of the channel diminished only to a minor extent from firm organisation and shrinking of the clot. Death from pulmonary gangrene occasionally occurs as the result of such clots, or portions of such dislodged, passing by the right cavities of the heart into the pulmonary vessels. The result of contraction of a limited vascular area of the cortex upon neighbouring territories must, we opine for the present, be a moot point; but we cannot fail to regard it as highly probable that any such limited spasm must tell in an exactly reversed vascular state of neighbouring cortical and subcortical tracts. The cortical nutrient branches form, as we have learnt from M. Duret, an absolutely-terminal (non-anastomotic) system of vessels, and (counter to the view of Heubner) the larger branches of the pia also map-out individualised and but feebly inter-communicating territories. Hence, we have reason to infer that each terminal system is the representative of a neuro-vascular autonomy; and that limited spasms of such a system, whilst raising the blood-pressure *generally* throughout the periphery, also cause increased flow to *neighbouring cortical realms*. How extremely delicate is the adjustment so affected is obvious from the researches of Mosso, who remarked that in the case of his patient, when asleep, the slightest sound, such as the tick of a watch, or a spoken word, short of awakening the sleeper, invariably caused increased vascularity of the brain, with a corresponding fall of blood-pressure in the arm, as registered by the plethysmograph.

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* "The common thickening of the membranes over the upper surface of the brain, increasing towards the longitudinal sinus, is explicable by the mechanical congestion that must be favoured there, through the current of blood from the cerebral veins entering the longitudinal sinus against the course of the stream within the sinus. The check so caused to the entering stream will have most effect on the part of the stream that is near the vein wall, for this is weaker than the current in the middle of the vein, but this parietal layer of the stream receives the blood from the parts near the sinus, and hence these will feel the check more than the distant parts, and will tend to be held all one's life in a state of mechanical congestion of mild degree" (*Lectures on Analytical Pathology*. Moxon).

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* "Pathological Anatomy," *Sydenham Soc.*, vol. iv., pp. 372-3.

† See also a similar case reported in the *Lancet* for January 11, 1879, by Dr. Coupland.

Such an observation gives us a graphic illustration of what is continually occurring, during the normal active processes in our conscious moments, as the mere result of sensory excitations alone.

A bright red blush irregularly distributed in patches is often observed in the cortex of those dying insane, a rosy-tinted mottling, stippled here and there with the orifices of larger vessels cut across, and defining (in most cases very accurately) the limits of certain independent vascular tracts or plexuses. It is more frequently an accompaniment of the more acute forms of insanity, and we hesitate to attach to it any further importance than as indicating the severity of the late functional disturbance. Certain it is that this appearance is not necessarily correlated with any obvious structural change in the part; nevertheless, it is a witness to the storm which has swept past. We have suggested elsewhere* that, "the last act of arterial contraction, in which the smaller arterioles have failed to empty themselves into the venous system, may in part explain this appearance," and have also noted how "this blotchy red aspect of the cortex reappears very frequently in the medulla in similar cases;" and we still regard it as probably so explained, the failure to contract being evidence of the paretic state of the vessel, whilst the effect of limited spasms would from our former remarks be still more likely to issue in this blotchy mottling of the cortex. The same remarks apply to the rosy vascular zones which so frequently present themselves along the junction of the white and grey matter. This and the fourth layer are usually the sites of the rosy discoloration now alluded to; and this coincides with the results of imperfect injections of the cortex, which indicate that the **long straight vessels** and their **horizontal nexus** on the confines of the grey are the most readily filled; next, the plexus around the cells of the fourth and fifth layers; and lastly, the vascular plexus in the third and the first layers respectively. We have alluded to this ready filling of these straight vessels of the medulla as a sort of safety-valve action for relieving the cortex from undue engorgement. The rosy mottling of the medulla is again a frequent accompaniment of the foregoing signs where such cerebral excitement has preceded death, or where epilepsy has terminated fatally in the "status;" and in such cases it is interesting to note the comparative paucity of **puncta vasculosa** which undoubtedly (as Niemeyer has stated) form a most unsafe criterion to accept of congested states of the brain.† Since, then, the appearances above detailed are occasionally the sequence of other than morbid states, how are we to deal with their significance as morbid signs? The reply is, in the presence of *minute extravasations*, in the *coarseness* or evident *disease of the small*

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* *The Human Brain: Methods of Examination*, p. 52.

† *Text-book of Medicine*, Niemeyer.

vessels, in the existence of much *œdema* of the tissues, in the altered *consistence* and *specific gravity* of the tissues; all of which afford indications of value ere we resort to more minute histological examination.

Increased vascularity is by no means the more frequent appearance found in fatal cases of insanity—in *acutely-maniacal* conditions, and especially in *general paralysis*, it is often observed; but, by far the larger number of cases afford evidence of poverty of blood in the brain and general malnutrition. Thus uniform pallor prevailed as a noteworthy feature in 841 cases out of a total of 1565 autopsies, or considerably over one-half (53·7 per cent.). What was stated respecting the independence of vascular regions of the brain as regards both the terminal arterioles and the larger areas mapped-off by the distribution of three main arteries of the cerebrum, is illustrated by the anæmic states of the cortex, even as we found it was by states of hyperæmia. We find conditions of patchy pallor mottling the cortex, or a uniformly diffused pallor, or, not by any means infrequent in cases of *melancholia*, a notable pallor of the carotid areas associated with fulness of vessels both of the white and grey substance in the vertebral system (posterior cerebral). By far the more extreme forms of anæmia met with in the insane are cases of chronic phthisis, unfortunately so frequent in asylums. The pallor is a most striking feature, and is due, of course, to general bloodlessness throughout the system. So also as the result of severe hæmorrhage, as in the *post-partum* hæmorrhage which may usher in puerperal insanity, this condition of blanching will be found present. Intracranial pressure, again, such as results from sanguineous apoplexy, the presence of adventitious growths, or excessive development of interstitial connective (*neuroglia*) may all lead to notable anæmia by forcible exclusion of blood from the cerebral vessels; but, here the blanched aspect of the surface is associated with so much flattening of the convolutions that the conditions are at once appreciated.

Inflammation.—Acute cerebritis as a **diffuse affection** of the brain we have had no experience of amongst the insane; on the other hand, as a **localised** condition due to focal lesion, it is by no means uncommon, whilst a chronic meningo-cerebritis is also of very frequent occurrence. To take the more acute process first, we usually find it as the result of an embolon or thrombus of a cerebral blood-vessel, which has given rise to much punctiform hæmorrhage around, or to hæmorrhagic foci in the cortex or ganglia; or, again, to the presence of new growths, such as carcinoma or tubercle, which are frequently surrounded by a zone of red inflammatory softening, beyond which extends a further non-inflammatory zone of simple white or yellow softening. The cerebral tissue so involved is swollen, distinctly œdematous, and variable in its consistence up to the extreme degree of diffluence; it is usually of bright pink hue, with streaky or punctated hæmorrhages scattered throughout its texture.

The inflamed tissue may show little or no discoloration, in fact this is very frequently the case; but in all instances we find the presence of inflammatory exudates modifying the appearance and textural continuity of the part, presenting compound granule-cells, nuclei, leucocytes, broken-down nerve-structures and pigment; whilst the specific gravity will be invariably augmented by the inflammatory exudation present. In the immediate neighbourhood of such inflammatory patches we often find the tissue in a state of white or yellow softening, non-inflammatory in character; in fact, due to interference with the nutrient supply of the part, to plugging of the minute vessels, or to direct pressure of the swollen œdematous tissue around the inflamed focus. There is no causal connection betwixt such states of inflammatory softening and insanity; they occur as accidents in the course of certain vascular diseases associated with mental disease, and, therefore, it is necessary to allude to them here. Far otherwise is it with the chronic inflammation of brain and membranes which is intimately related to the insidious and fatal malady, general paralysis. In this form of chronic meningo-cerebritis the inflammatory changes begin, probably, at several different points, spreading from one convolution to another, until in many cases the whole cerebral mantle is involved, with the exception of the occipital gyri, which almost invariably escape implication. The progress of inflammatory activity is usually most marked in the frontal regions in both hemispheres, and less advanced in the parietal. The cortex is much thinned in the fronto-parietal region, and of very variable colour, frequently exhibiting the irregular mottled aspect from pinkish discolorations or congested patches, but also quite as often pale grey, or of a uniform dirty grey hue with but poorly-defined lamination. The arterioles of the cortex are frequently and notably coarse and engorged. The substance of the cortex is much reduced in consistence, and œdematous; the whole brain is softened, and has an ill-nourished look; in fact, apart from the firm inflammatory adhesion of the opaque and thickened membranes, the naked-eye appearances of the cortex are not unlike an extremely ill-nourished and atrophied brain in old age, presenting in itself no characteristic indications of the extensive inflammatory changes which it has undergone.

Softening.—Out of 853 cases of insanity proving fatal, 390 afforded instances of an increased consistence of brain or one of average firmness; the remainder were noted as having a diminished consistence throughout. The actual figures were as follows:—

Increased consistence throughout	. . . . .	98 cases.
Firm consistence ,,	. . . . .	110 ,,
Fair or average consistence ,,	. . . . .	182 ,,
Diminished consistence ,,	. . . . .	463 ,,

Roughly speaking, therefore, close upon one-half (or 45·7 per cent.) were of normal consistence or *above* the usual degree of firmness—the remaining 54·2 per cent. being softened, either as the result of disease or of *post-mortem* change. As a fact, however, a state of general reduced consistence, apart from any putrefactive process, prevails in a large percentage of cases; and this is accounted for by the large proportion of cases of senile atrophy, of general paralysis, and of organic brain-disease (the result of diseased arteries) which accumulate in our asylums and form so large a fraction of the fatal cases. The *general* diminution in cerebral consistence may be due to œdema of its texture, as the grey matter has notable hygrometric powers; it may be due to disintegration of structure from the fatty degeneration of senility, or from extensive vascular disease restricting its nutrient supply, or it may be the result of inflammatory processes. In all cases we observe that the vascular system is largely involved. The œdema is first established by the undue engorgement of vessels which thus relieve themselves (not only in congestive and inflammatory processes, but also in the atrophy resulting in the so-called *hydrops ex vacuo*); the fatty disintegration of senile brain is invariably associated with, and greatly furthered by, diseased arterial tunics; and, lastly, the inflammatory processes, which are of a chronic diffuse nature, are, we believe, in themselves vascular in their origin. Hence we see how large a section of the insane show indications of defective nutrition in the central nervous system, and derangements of its blood supply; yet acute or recent insanity affords few and far less pronounced signs of such impairment. It is in the chronic stage of insanity that obvious structural changes indicate to us the serious nature of the nutritive failure. In instances of general reduction in the consistence of the brain, the organ fails on removal to maintain its erect position; it falls apart at the commissural junctions, the diverging hemispheres tending by the mere effect of gravity to tear the latter asunder, especially as these commissures are themselves unduly soft. The hemispheres have lost their plump contour; the convolutions may have undergone considerable atrophy, and their widely-gaping sulci may enclose much serous fluid; whilst the whole brain feels flabby to the touch, and devoid of its normal compact aspect, as well as of the firm and resilient feel of healthy structure. In the ventricles we often find considerable serous fluid unduly distending these cavities, whose walls have a macerated aspect, and are undergoing rapid solutions of continuity. The white substance may have a glairy, brilliant aspect, be much softened in texture, pit on pressure, whilst few or no puncta vasculosa appear, the vessels being compressed by the swollen œdematous structure around; or it may, on the other hand, have a dull, lack-lustre surface, mottled with diffused congested zones, stained with hæmatine, and presenting numerous coarse and bristly vessels.

In extreme cases of white softening, however, the brain-substance may be completely diffluent here and there, and present to the touch a semi-fluctuating feel, breaking up readily upon manipulation; its central parts—the fornix, septum pellucidum, and commissure—being more or less wholly disintegrated. Such localised softenings superadded to the more generally diffused form above described, are due to the association of plugging of the cerebral blood-vessels by thrombus or embolon. The substance of the brain thus implicated may be reduced to a soft, pappy, cream-like fluid; or the *almost* diffluent medulla may wash away in this form by the impact of a gentle stream of water. Localised softenings, as the result of thrombosis or embolism, frequently illustrate to us the regional autonomy of the cerebral blood-vessels by invading only the district of supply of one of the principal branches of the three large vessels of the cerebrum; and thus we may have lesions of one of three districts of the interior, or of the posterior cerebral, or one of the four districts of the middle cerebral, exclusively, or variously associated.

In this connection undoubtedly the area of supply of the **middle cerebral** is far the more frequently affected; next to this, but with far less frequency, the posterior cerebral areas suffer; and least frequently of all, the areas supplied by the **anterior cerebral.** The relative implication of the various branches, or rather their territories of distribution, are for the middle cerebral or Sylvian trunk as follows (the more frequently implicated branches in order of precedence):—Parieto-sphenoidal, ascending frontal, ascending parietal, external and inferior frontal branches. For the posterior cerebral the order of precedence is—first, the occipital branch, and, far less frequently, the two anterior branches to the uncinate and fusiform gyri. Again, for the anterior cerebral the order of precedence in morbid implication is—the middle and external frontal branch, the anterior and internal frontal, and, least frequently, the posterior and internal branch. This may be more clearly represented in the following tabulation—

Anterior Cerebral.	Middle Cerebral.	Posterior Cerebral.
Middle and internal frontal artery.	Parieto-sphenoidal artery	Occipital artery.
Anterior ,, ,, ,,	Ascending frontal ,,	Anterior and posterior temporal artery.
Posterior ,, ,, ,,	Ascending parietal ,,	
	External and inferior frontal ,,	

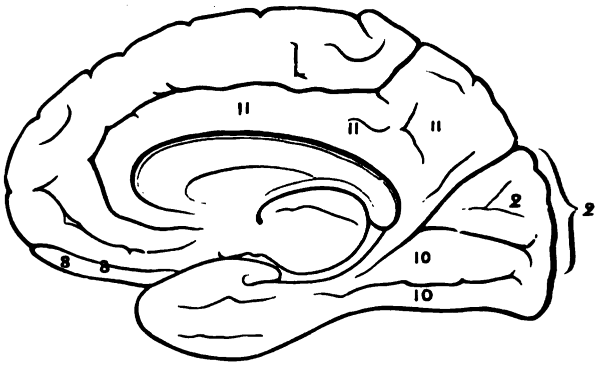

Fig. 21.—Illustrative of the more frequent site of localised softenings in order of precedence.

In 149 cases of localised lesions, the convolutions most frequently affected, and medullated centrum would run as follows in like order of precedence :—

1. Upper temporo-sphenoidal gyrus.
2. Occipital and cuneate ,,
3. Ascending frontal ,,
4. Postero-parietal ,,
5. Centrum ovale = not defining as to anterior or middle frontal territory.
6. Annectants.
7. Angular and supramarginal.
8. Orbital.
9. Insula and operculum.
10. Fusiform and uncinate.
11. Gyrus fornicatus and quadrate.

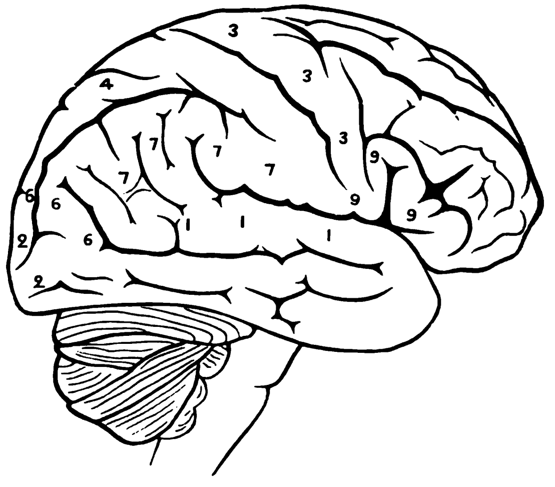

Fig. 22.—Illustrative of the more frequent site of localised softenings in order of precedence.

A further analysis of 166 cases of localised or focal softenings (due either to thrombosis, embolism, or to hæmorrhage) in the substance of the basal ganglia, and their medullated capsules indicate the respective proclivity to such lesions in the insane to be as follows:—The *left* hemisphere is in all instances of ganglionic lesion, *slightly* more prone to implication; the intraventricular nucleus is far the more frequently affected, the optic thalamus comes next in frequency, but the proportion does not rise beyond two-thirds the latter; the extraventricular or lenticular nucleus is somewhat less frequently involved than the optic thalamus; lastly, the two capsules, external and internal, are far less often implicated, and of these the inner shares the greatest immunity.

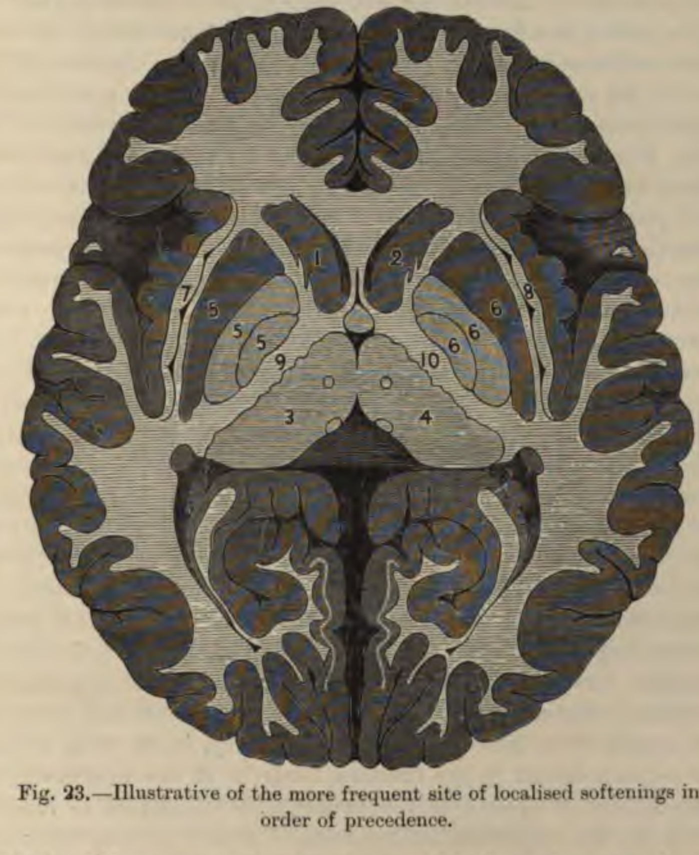

Fig. 23.—Illustrative of the more frequent site of localised softenings in order of precedence.

Of the 166 cases of softening from which these data are obtained, thirty-four were instances of hæmorrhage, and the remaining number were the results of clot, usually thrombosis. The results agree, therefore, in every particular with those arrived at from a study of the locality of cerebral hæmorrhage, for it has been shown by Andral and Durand-Fardel, that the corpora striata are more frequently the site of hæmorrhage than the thalami; and Charcot expresses his opinion that next to the opto-striate bodies, the claustrum is the more frequent site of lesion.* Whatever be the origin of the cerebral hæmorrhages met with in the insane (whether the result of a periarteritis or an endarteritis), this we can safely affirm, that the same systems of vessels have the same relative liability to suffer in cases of softening, following occlusion from thrombosis, as in cases of hæmorrhagic foci.

Atrophy.—Wasting of the grey and white medullated structures of the brain is of very frequent occurrence in insanity, but it is as a sequel to the acute forms; and, in the chronic forms of insanity, such atrophy is seen to invade these structures to an extensive degree. It may be *general* throughout the cerebral hemispheres, whilst the basal ganglia and mesencephalon escape implication; but, occasionally, the whole of the intracranial ganglia are involved. On the other hand, it may be *localised* or *partial*, when it may implicate any region of the brain. It may be *rapidly* induced as the result of an inflammatory process; or it may be of *extremely slow and insidious progress*; or the steady progressive dissolution implicating the whole cerebrum, which distinguishes the atrophy of premature senility. The intimate structure of the central nervous system would indicate peculiar relationships as established betwixt the individual elements which must be fully recognised ere we clearly appreciate the significance of these various forms of atrophic change. Let us take as illustration of our remarks the district supplied by one of the terminal cortical arteries. Now, we affirm that the autonomy of this department demands a mutual sympathy betwixt all the constituent elements of the same; in other words, action and reaction is so established between them that any derangement in the functional activity of the one *must*, of necessity, affect the other. In fact, the more *highly differentiated* the structural parts of a tissue become, the more *dependent* also do they mutually become. It matters little for this terminal arterial territory if a distant branch (however large) off the same trunk be plugged, it still maintains its autonomy; but it matters very much if this minute branch itself be obstructed. The nerve-cell is dependent upon the terminal artery for a due supply of its nutrient plasma; the artery, in its turn, is regulated as to its calibre by the functional activity of the nerve-cell; the lymph-connective system of the neuroglia is stimulated to renewed activity by the accumulating products of nerve disintegration; the nervous elements depend upon this continuous removal of effete material for their normal storage and discharge of energy; and so, in like manner, the connective and vascular elements are mutually dependent. In no organ of the body is this mutual dependence of parts so exquisitely elaborated as in the brain and, *a fortiori*, the cerebral cortex. Terminal vessels exist elsewhere, as in the spleen, kidney, and lung; but the presence of the nervous element establishes a much more complete mutual dependence of parts. This inter-dependence of the structural elements of the cortex, due to its terminal system of arteries, is of primary importance to us in correctly appreciating the morbid appearances presented in insanity. Another factor, however, must be invariably considered with respect to all morbid lesions of the cortex, and that is the sympathy betwixt *distant* territories which are functionally associated in their activities, and structurally linked together by "association" fibres. The former condition—the inter-dependence of parts in terminal systems—was the direct outcome of elaborate differentiation; the latter condition of sympathy betwixt distant territories is established by an equally elaborate structural integration.

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* *Localisations in Cerebral Diseases.*

Keeping these facts in view, it becomes obvious that much obscurity naturally overshadows many pathological processes in the cortex cerebri, despite the prominence of the morbid changes presented. No one element of the tissue of the cortex can suffer materially, without rapidly disturbing the nutritive equilibrium consistent with the health of the territory to which it is attached; hence it often becomes a question whether changes observed in the nerve-cells are evidence of primary implications, or whether they are secondarily induced through a disturbance in the circulation of the district, or impairment of the lymphatic functions of the cortex, in a blood crasis, or other cause; or, again, as in the medulla, whether a sclerosic change with atrophy of nerve tubuli is primary parenchymatous (originating in the nervous element), or interstitial (spreading from the neuroglia to the latter). There is every reason to believe that in the nervous centres both parenchymatous and interstitial change may occur as the primary fact; that the nerve-cell, for instance, may be stamped with a morbid instability wholly independent of any *ab extra* agency, and this as an inherited or as an acquired condition; nor is it unreasonable to suppose that the changes in the nerve-cell in physiological senescence are *initiated* apart from any nutritive anomalies and blood vascular changes, being simply the expression of the expiration of its fixed term of existence.

The very general atrophy of the cerebral cortex occurring in pathological sequence is often, but by no means invariably, associated with a degeneration of its nutrient vessels, and when these vessels are involved it is to a very varying degree. Yet what is invariably found is the degeneration of the nerve-cells which, in any appreciable degree of atrophy, are extensively and very notably implicated. We have here, in fact, what may be regarded as a true parenchymatous degeneration; the primary change is initiated in the nerve-cell.

Other forms of atrophy, usually more limited in distribution, occur as the result of *over-action* of nervous centres; in such cases the element which chiefly assumes the morbid *rôle* is the connective matrix or neuroglia, although the primary incitant was undoubtedly nervous. Illustrations are afforded in the case of alcoholism, where repeated over-stimulation of nervous elements and the waste and effete material so produced, demand from the lymph-connective system more than its capabilities can accomplish; tne result is a temporary hypertrophy of this tissue, the multiplication of its active elements (*phagocytes*) followed by their fibrillation, and the eventual atrophy due to the encroachments of the connective upon the nervous elements. A different illustration is afforded by cases of epileptic insanity, for here again over-action leads to degeneration and atrophy of nerve-cells through the medium of an encroaching connective; the conditions of the epileptic are, however, by no means parallel to the alcoholic, and we find that, in lieu of actual atrophy of the brain-mass, there is often hypertrophy and augmented density due to the inordinate growth of the connective element. Instances of premature senility (or what used to be called atrophia cerebri precox) are illustrative of this form of atrophy from over-activity of nervous centres. Long continued or oft repeated excitement induces a similar state of atrophy, as seen in most fatal cases of chronic insanity.

A frequent cause of localised shrinking of the brain-substance is the destruction and cicatricial condensation of tissue in hæmorrhagic foci; or, again, as the result of inflammatory destruction of nervous tissue, whether arising centrally or spreading inwards from the meninges. Either of these affections occurring in *infancy* will almost certainly, owing to the principle enunciated above, retard the development of distant parts, so that in adult life the brain will exhibit great disparity in its two hemispheres, and in different regions of the affected hemisphere. Such cases also illustrate the crossed connection betwixt cerebrum and cerebellum, and between the cerebellum and the opposite olivary body, such as have been indicated by Van-der-Kolk and Meynert. As illustrative of this condition, may be mentioned the case of a paralytic idiot, the subject of right hemiplegia and epilepsy, who died at the West Riding Asylum, and in whom there was found atrophy, with sclerosis of the left cerebral hemisphere, associated with atrophy of the right lobe of the cerebellum (*Major*);* also the case of another patient at this asylum, in which a lesion of one hemisphere of the cerebellum, of the nature of an old hæmorrhagic cavity with dense sclerous walls implicating the corpus dentatum, was associated with degeneration of the olivary body of the opposite side (*Dudley*).*

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* See descriptive summary with illustrations of this case by Dr. H. C. Major, *Journ. Mental Sc.*, July, 1879.

The frequency of atrophy of the cerebral hemispheres in insanity, may be conclusively shown by the *post-mortem* statistics afforded at the West Riding Asylum. It is hereby shown that, out of a total of 1,565 fatal cases of all forms of insanity, as many as 1,055 (or 67·4 per cent.) presented evidence of cerebral atrophy; that the wasting was *general* throughout the hemisphere in 574 of these cases, although 261 also exhibited a *special* implication of certain areas, and that in 481 other cases partial or localised atrophy was observed. The fronto-parietal segment of the hemispheres enjoys least immunity from the atrophy incident to insanity, and we find atrophy confined to the frontal lobes in a large number of cases; in fact, in the proportion of three-fourths to one-fourth respectively. Of still more restricted areas the postero-parietal lobule appears most prone to a localised atrophy (forty cases); the central gyri ranking next to this lobule in the frequency of their implication (thirty-seven cases); the separate frontal gyri and Sylvian boundary (operculum) were thus affected in thirty-three and twenty-nine cases respectively; then followed the temporo-sphenoidal and the occipital gyri, the angular being implicated in but eight cases. The general results arrived at by a large series of weights of the brain in the insane, excluding cases of congenital defect, teach us that the lowest average weight due to atrophy is attained by instances of so-

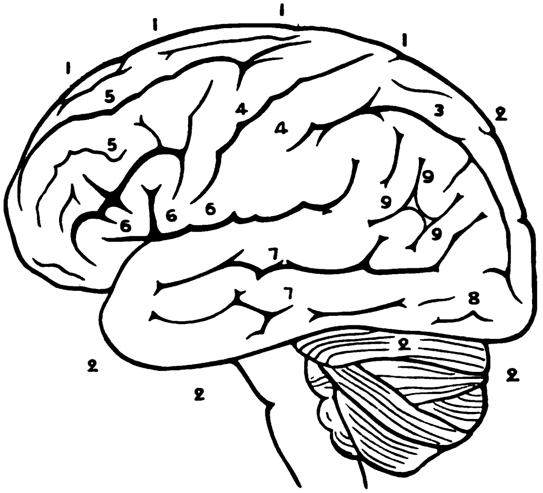

Fig. 24.—Illustrative of the sites of election of atrophy in order of precedence.

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* See report with illustrations by Dr. William Dudley, *Journ. Mental Sc.*, July, 1886; see also paper on "Atrophy and Sclerosis of the Cerebellum," by C. Hubert Bond, *Journ. Mental Sc.*, July, 1895.

called organic dementia (hæmorrhagic or ischæmic softenings); senile atrophy of the brain follows next; whilst general paralysis ranks third in order.

Miliary Sclerosis.—So frequently has the lesion, to which this term is applied, been recognised as occurring in the central nervous system of those dying insane, since it was first described and figured by Drs. Batty Tuke and Rutherford in 1868, that we feel diffident in suggesting a new name; although we differ from those observers in regarding its nature as that of genuine sclerosis or overgrowth of the connective element of the nervous tissues. That the morbid change hitherto recognised under this term must eventually be renamed we are confident; and that when its nature is fully understood its importance will gain for it much greater attention than it has hitherto received we are equally confident; meanwhile, we retain the name whereby it has been known for the past thirty years, as the least likely to mislead the student as to the change to which we refer. The morbid appearance presented by this lesion has been the subject of frequent examination by many observers, but little (if anything) has been added to the graphic account given by its discoverers.* Some, however, have gone the length of denying its pathological origin, asserting it to be a *post-mortem* change or an artificial product cleverly manufactured by the histologist's reagents: amyloid, colloid bodies, and "miliary sclerosis," have all in their turn been explained away by some as the results of alcohol or other reagents. Unfortunately for this theory, however, all such changes are to be found in the *perfectly-fresh brain* before any reagent has been applied, and it requires but a short experience in the fresh preparation of nervous structures amongst the insane to vindicate their pathological import. Dr. Batty Tuke's description of the fully-developed lesion is as follows:—

> "As a rule, the spots are unilocular, occasionally bilocular, and in rare instances multilocular; but whatever their condition in this respect is, they possess the same internal characteristics. A thin section prepared in chromic acid viewed by the naked eye shows a number of opaque spots irregularly distributed over the surface of the white matter; they are best seen in a tinted section, as they are not colourable by carmine. When magnified by a low power they have a somewhat luminous pearly lustre, and when magnified 250 and 800 diameters linear, they are seen to consist of molecular material, with a stroma of exceedingly delicate colourless fibrils. They possess a well-defined outline, and the neighbouring nerve-fibres and blood-vessels are pushed aside, and curve round them. In well-advanced cases the plasm seems denser at the circumference of the spots than at their centre, and a degree of absorption of the contiguous nerve-fibres is evident; this solution of continuity is only noticeable at the point where the lateral expansion is greatest. The

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* See especially Dr. Batty Tuke's article on the "Morbid Histology of the Brain and Spinal Cord in the Insane," *Brit. and For. Medico-Chir. Review*, July, 1873; also Dr. Kesteven's paper in the *Brit. and For. Medico-Chir. Review*, April, 1869.

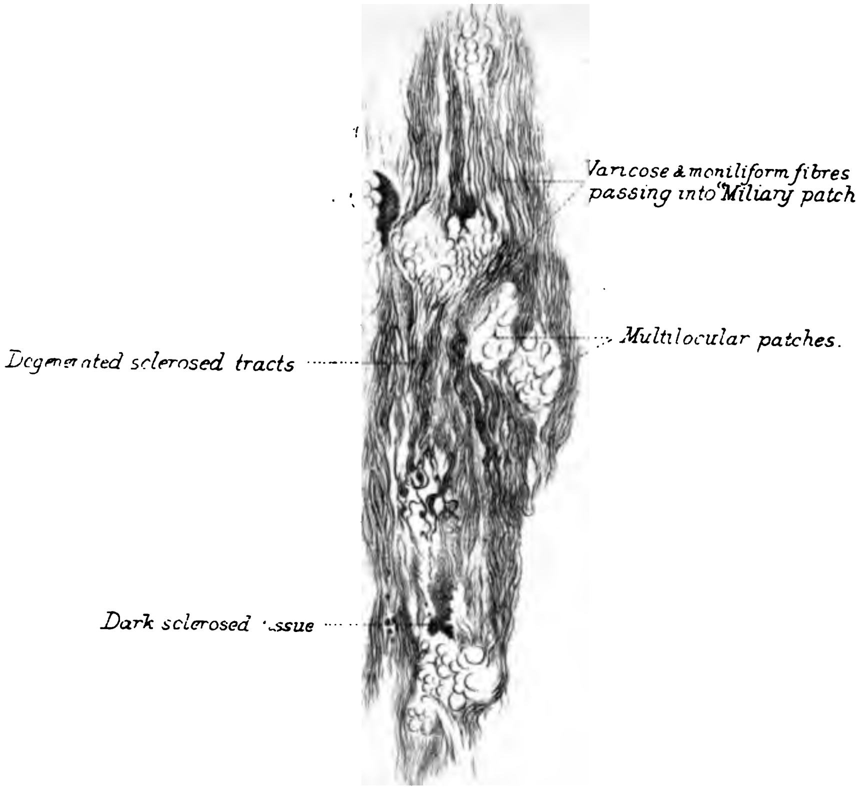
Varicose & moniliform fibres passing into "Miliary patch."
Multilocular patches.
Degenerated sclerosed tracts
Dark sclerosed tissue

Fig. 1

Degeneration of medullated fibre in lateral columns of Spinal cord forming so-called "Miliary Sclerosis" as seen under low power objective.

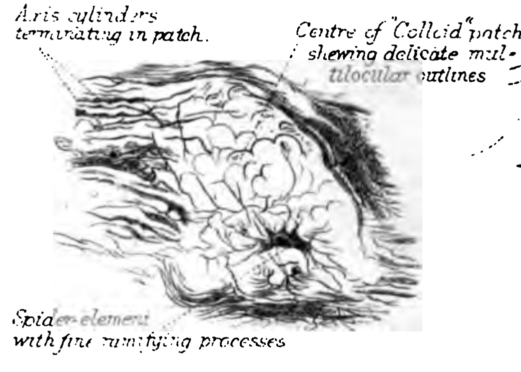
Axis cylinders terminating in patch.
Centre of "Colloid" patch shewing delicate multilocular outlines
Spider element with fine ramifying processes

Fig. 2

"Colloid" patch resulting from Degeneration of medullated fibres of cord.

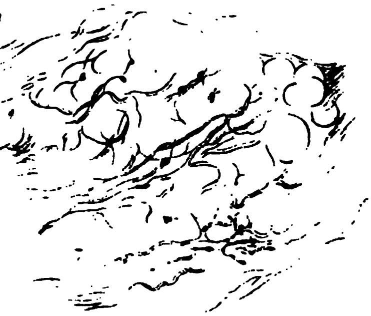

Fig 3.

"Colloid" patch more highly magnified shewing outline of multilocular material & fine stroma of elastic fibre. x 350

Bale & Danielsson, Ld.

spots are generally colourless, but in some instances they are of a yellowish-green tint, which may be attributable to chromic acid. They vary much in size; unilocular patches are $\frac{1}{50}$th of an inch to $\frac{1}{100}$th of an inch in diameter, the multilocular from over $\frac{1}{300}$th to $\frac{1}{500}$th of an inch. As many as eleven locules have been noticed in one patch, separated one from the other by fine trabeculæ of nervous tissue."*

The favourite sites of election for the development of these morbid appearances seem to be the white matter of the cerebrum, the pons and medulla, and the lateral columns of the spinal cord. In the last position it can be studied to best advantage, although it is far more generally met with in the medulla of the cerebral hemispheres. When its occurrence is noted in the brain, we find that on holding up a stained section slightly aslant to the light, numerous little bright pellucid points appear scattered through the medullated structure, and just perceptible to the naked eye; they are also seen by reflected light, but not by direct transmitted light. Under a low power each of these brilliant points is resolved into a distinctly lobulated patch some 20 μ to 50 μ in diameter, their colourless aspect strongly contrasting with the stained tissues in which they are imbedded. At first sight they look like a number of unequal sized droplets of a fluid of somewhat dense unctuous consistence, which have incompletely fused with each other; and which, with somewhat oblique light, stand out in bold relief with opalescent or frosted pearly lustre. By direct light they appear more translucent, and often seem like unequally lobulated cavities (in some cases actually being so, the contents having fallen out during the preparation of the specimen). They present, moreover, three suggestive features which have a direct bearing on the question of their pathogenesis.

(*a*) They are distinctly limited to the **white medullated structure** of the brain; and, where they approach the grey cortex of the convolutions, they, in most cases, *abruptly terminate*, the naked-eye suffices to elicit this limitation. When they do invade the cortex, as very rarely occurs, it is only in its lowermost zone, and then strictly along the line of the large medullated radiations.

(*b*) The perivascular nuclei frequently exhibit abundant proliferation, and granular hæmatoidin masses freely cover the sheath of the vessel.

(*c*) The condition is at a certain stage invariably associated with an increase of the so-called spider-cells or Deiter's corpuscles.

If the spinal cord rather than the brain be the subject of our scrutiny, we find the lesion presents still more prominent and obtrusive indications of its presence. Its demonstration is not only facilitated by certain features here presented, but the essential nature of the change also becomes clearly evident. On examining, by unaided vision, a stained, transverse section of a spinal cord so affected, we

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* *Loc. cit.*, p. 205.

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find a dark-stained area undoubtedly sclerosic in nature) of one or both lateral columns *apparently* riddled by numerous minute apertures; in reality, they are *not* apertures, but minute foci readily transmitting light, owing to their altered tissue and resistance to all staining reagents. We have here, in fact, a system-disease of the cord—a lateral sclerosis with certain peculiar morbid features superadded.

At the site of these apparent apertures the microscope reveals colourless translucent patches, irregular in contour, usually more or less lobulated, and frequently showing in their midst indications of varicose medullated fibres; reproducing in other respects the appearances above described in the miliary patches of the brain. Around such unstained areas the tissue is always condensed and most deeply stained, and the nerve-elements are much wasted or completely replaced by sclerosed tissue (*Pl.* xiv., *fig.* 1). Far more instructive specimens, however, are obtained from *longitudinal* sections through the diseased columns. The morbid product is then seen to be aggregated in oval or elongated elliptic patches measuring 139 μ to 186 μ in length by 40 μ to 70 μ in breadth: and in many cases the morbid material has dropped out, leaving only an irregular opening, the boundaries of which are fibrillated and never clean-cut or punched-out through the tissues, as are certain channels of morbid origin found occasionally at these sites. The appearance at once suggests to the mind the forcible extravasations at numerous points of a coagulable material which has driven the textural elements asunder before it: a suggestion further favoured by the almost invariable presence of a blood-vessel (often of considerable magnitude) running in close proximity to, or even appearing to lose itself in, the morbid focus. The morbid material is seen to consist of a congeries of oval or spherical segments of delicate and indistinct outline, the sole indication of which is often the gentle curve of a connective fibre passing over it (*Pl.* xiv., *fig.* 2). Usually pellucid, it may be found slightly opalescent, whilst overlying and passing between its segments is an extremely fine plexus of fibrils. Some of the fibres branch dichotomously, and have the appearance of veritable elastic fibres; others arise from delicate spider-cells which are numerously scattered around the confines of the diseased patch (*Pl.* xiv., *fig.* 3). Around their lobulated contour we find a condensation of tissue, the nerve-fibres atrophied or absent, and the sclerous tissue and blood-vessels closely packed and curving round the mass. But the more important point to note is that all the medullated fibres, in a line with the diseased tract, are in a condition of advanced disease, and end directly in this morbid focus; extreme varicosity with segmentation of the medullated sheath is apparent. These medullated fibres, as they approach the diseased focus, are, in many cases, seen to be regularly moniliform, segmentation of the white matter of Schwann having proceeded so far that spherical masses of medulla are strung upon the axis-cylinder like beads upon a string; at times the retraction of the segmented portion is not so great, and an irregularly varicose aspect ensues; whilst, in other cases, large pyriform masses of medulla are seen, the axis-cylinder extending like a stalk from its narrow end (*Pl.* xv.). Beside these varicose fibres lie naked axis-cylinders wholly devoid of an investing medulla, and swollen beyond their natural dimensions.

The spherical masses of medulla exhibit a series of progressive changes towards disintegration. The early stage is represented by the simple spheroid or pear-shaped mass, perfectly clear and translucent, with, perhaps, a faint indication of the axis-cylinder running through its centre or displaced laterally. The next stage presents a slightly-frosted clouding of its interior, followed later on by the formation of numerous extremely minute granules within. In the third stage the spheroid becomes not only full of these granules (apparently fatty in nature), but its whole mass takes up a faint staining of aniline or hæmatoxylin dye, whereby it is distinctly contrasted with the clear spheroids of an earlier stage which remain unstained (*Pl.* xv.).

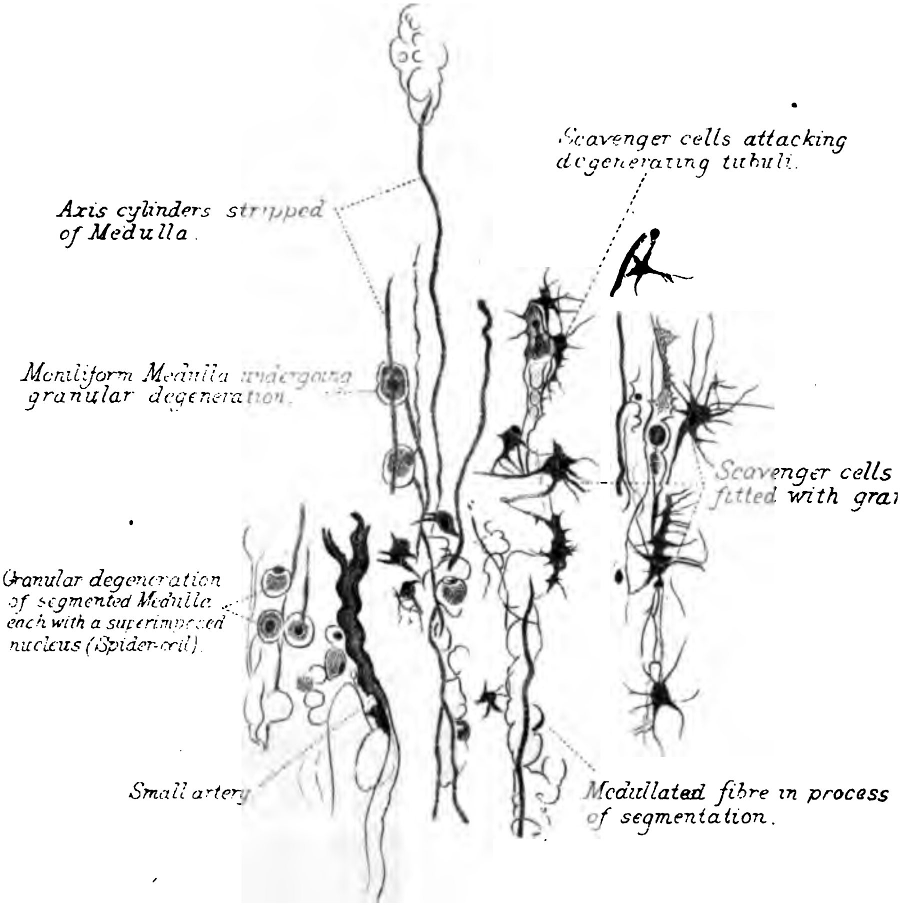
In the immediate neighbourhood of these segmented medullated fibres careful examination reveals a vast number of spider-cells; so delicately pellucid are these bodies, and so faintly do they take up the staining reagent, that they are readily mistaken for free nuclei, since their nucleus is always deeply stained, and this error is not unfrequently committed. At the height of the morbid activity, however, the nature of these elements undergoes a remarkable change; they then stain well, even to their ultimate ramifications, and they apply themselves so well to the task of removing disintegrative material arising from the nerve-tissue that their interior soon becomes filled with the minute granules which we have referred to as clouding the medullary spheroids (*Pl.* xiv., *fig.* 2, *Pl.* xv.). These lymph-connective elements thrive abundantly at the expense of the nerve-tissue and enlarge greatly in bulk, whilst their extensive ramifications pervade its structure in all directions. Along the course of such degenerating medullary fibres the bead-like segmentation often produces a close resemblance to a series of large cells, a resemblance which becomes most striking by the occurrence of what looks like a fair-sized nucleus occupying the centre of each sphere; undoubtedly they have been frequently described as cells, an error not to be wondered at since few reagents display well their real nature; carmine and hæmatoxylin help to falsify the appearance, but aniline blue-black with bright illumination seems best adapted for exhibiting their real constitution. With other dyes they might readily pass for nuclei arranged along the nerve-fibre at each of its moniliform segments, did we not know that the medullated fibres of the centric nervous system (unlike the peripheral nerves) are devoid both of nuclei and of Schwann's sheath, being, in fact, not made up of inter-annular segments. Properly-prepared sections, however, show that this apparent nucleus does not occupy the interior, but lies upon the exterior of the sphere, and is in reality the nucleus of a young **spider-cell,** surrounded by a little granular protoplasm from which delicate branches radiate and clasp the spherule of myelin (*Pl.* xv.). Being closely appressed to the spheroid, it looks like a protruding nucleus; and the regularity with which a whole series of such moniliform enlargements in a line with each other show these aggressive cells, would appear to indicate a remarkable morbid affinity. It is also to be noted that where these nucleated spider-cells apply themselves, the medullated spheres have all undergone a **granular change** and admit of **staining;** where the myelin-droplets remain clear, homogeneous, and unstained, these nucleated cells do not present themselves. On the other hand, these scavenger-cells appear abundantly in the dense sclerosic tissue immediately surrounding a **miliary patch.**

The degenerative change which we have thus followed in the medullated fibres of the spinal cord is recognisable as an all-important feature in the white medullated strands of the cerebral convolutions in chronic alcoholism, and especially in senile atrophy of the brain; the same activity of the lymph-connective system prevails in these cases, and (as we shall see when treating of these affections) spider-cells accumulate around the disintegrating nerve-fibre. In the cortex of the brain these spider-cells are often found (during the disintegrative stages of disease) to contain small masses of deeper stained material apparently derived from the neighbouring nerve-cells (*Pl.* xxiii., *fig.* 3). Eventually the fibrillation so resulting entirely replaces the nervous tissue, so that deep-stained tracts consisting of sclerous tissue only are seen here and there at the more advanced sites of disease.

Thus, in the immediate neighbourhood of these patches of *miliary sclerosis*, we find the nervous tissue in a state of **parenchymatous degeneration,** which, resulting in destruction and atrophy of the essential elements, becomes the site of a **genuine sclerosis.** What relationship exists between the unstained patches of miliary sclerosis and the condition of parenchymatous degeneration around?

We have already referred to the invariable presence of a fair-sized blood-vessel lying in direct contact with these patches of *miliary* degeneration. If these be closely examined we find reason for believing that the coats of the vessel are involved by extension in the morbid process, the coats are unduly thickened, the perivascular nuclei have

Plate XV.


Scavenger cells attacking degenerating tubuli.
Axis cylinders stripped of Medulla.
Moniliform Medulla undergoing granular degeneration.
Scavenger cells fitted with grai
Granular degeneration of segmented Medulla each with a superimposed nucleus (Spider-cell).
Small artery
Medullated fibre in process of segmentation.

Degeneration of Nerve-fibres of lateral columns of Spinal cord in so-called "Colloid degeneration" of these tracts.

Bale & Danielsson, J

undergone great proliferation, the vessels are much contorted, and very frequently occluded. We would suggest that the patch of miliary degeneration may be directly due to this implication of a neighbouring blood-vessel, by the exudation from the vessel inducing such swelling of the myelin as to rupture the delicate investing albuminous sheath, or possibly by a direct action upon the latter. The patch undoubtedly consists of altered myelin exuded in droplets from the medullated tubes and coalescing more or less completely—the axis-cylinders forced aside with the neighbouring tissues, or undergoing complete solution of continuity. In a *large proportion* there can be very little doubt that disruption of the axis-cylinder occurs, judging from the appearances presented by the section of the miliary spot. The skeleton framework of the structure, however, still remains in the form of a delicate plexus of elastic fibrils beautifully dissected-out by the process, and brought into relief upon the colourless spherules of myelin; with these there becomes blended ultimately a fine stroma of fibres arising from the spider-cells around. The latter condition occasionally proceeds to an excessive extent in similarly degenerative foci in the cerebrum and cerebellum, when we meet with isolated tufts of delicate interlacing fibres forming dense meshworks (beautifully revealed by aniline dyes in the fresh brain), which are devoid of nervous structure and of all cellular elements alike. In other instances the small nodule falls out during preparation, or (as Dr. Batty Tuke observes) may be picked out with the point of a knife from the hardened brain. The naked-eye appearance of these formations, as they present themselves *in situ* in a segment of the cord, is that of just perceptible points perfectly white and opaque, and thus contrasted strongly with the deep chrome tint of the surrounding tissue. Removed by a pin-point to a glass slide, they are found to resist considerably the pressure applied to the cover glass, but upon the addition of a drop of bichromate of potash solution firm pressure resolves them into tolerably large spherical bodies. Osmic acid does not darken these morbid formations, but this fact might be anticipated from the change induced by the chrome salts. The effect of nitric acid and other reagents has been studied by Dr. Rutherford; the former renders the mass transparent, and subsequently resolves it into a number of colourless bodies about the size of a blood-corpuscle, apparently formed by the coalescence of droplets occasioned by the acid solution of the mass. By pressure on the cover glass these bodies become elongated and, eventually, removed entirely, leaving a delicate fibrous stroma of connective in their place. Strong sulphuric acid acts similarly. It will be seen from the above remarks that we regard these multiple lesions not as a primary sclerosic change, but as **accidents** occurring in the course of a **subacute inflammatory** or **degenerative change** in the medullated nerve tracts; and that when (during the progress of a parenchymatous inflammation of these structures) the tunics of an adjacent blood-vessel become involved, there we get the rupture of these globose masses in the nerve-tubuli and their coalescence in the patches of miliary deposit, which are most probably altered in constitution by the inflammatory effusion from the blood-vessel. That it is not an essential feature in the history of such inflammatory activity is sufficiently evidenced by its frequent absence; yet, it is our opinion that in the nervous tissues of the insane its occurrence is more frequent than is usually supposed.

It is well known how difficult of demonstration are the several degenerative and chronic inflammatory changes which occur in the medullated tracts of the brain in insanity, and how liable such changes are to be overlooked. The presence of such miliary spots, therefore, is one of very great interest and importance, as calling attention to the morbid state of the nerve-fibres in the immediate neighbourhood.[*]

Colloid Degeneration.—A very frequent lesion found in the brain of the insane is that which has been termed "colloid degeneration," a term applied to the presence of minute round or oval bodies, from 6 μ to 12 μ in diameter, which pervade the nervous structures occasionally in extraordinary numbers. The frequency of its occurrence in the brain and spinal system of the insane, its undoubtedly morbid origin, and the essential nature of the lesion indicate it as one of the most important conditions for our consideration in the morbid histology of insanity.

Some nine years ago we described as a frequent appearance in the nervous tissues of the insane certain peculiar morbid products, which, *although undoubtedly derived from the medullated nerve-fibre*, bore a striking resemblance to the so-called colloid bodies,* and we ventured to suggest their actual identity, but withheld any dogmatic statement of the case, until further observation had assured us that the usually received opinion of their constitution was fallacious. Repeated observations since this date fully confirm our former suggestion that these morbid products have been too hastily relegated to the chapter of diseases of the connective framework or neuroglia; and assure us, moreover, that the morbid bodies then described by us were identical in their nature with the "colloid" body. The name is unfortunate, since it assumes a colloid transformation of a connective cell similar to what occurs in the typical colloidal transformation of the epithelia of the thyroid gland, or the same change in the elements of new growths, and we feel convinced that in this *cellular* origin of the change the view is inaccurate. In *size* these bodies vary very considerably, from 6 to 12 μ in diameter, up to 40 μ—the former being the usual dimensions of those found in the cerebral convolutions, the latter those of the regions of large medullated fibres, such as the medulla oblongata. Dr. Batty Tuke gives their diameter at $\frac{1}{2000}$ to $\frac{1}{4000}$ of an inch, but this clearly applies to the minute colloid bodies of the cerebral gyri; he also notes their variability in size, quoting certain experiments of his own and Dr. M'Kendrick on the brain of pigeons in which colloid bodies were discovered of very minute size ($\frac{1}{5000}$ inch). As we have elsewhere stated, they vary in direct relation to the varying diameter of the medullated nerve-tracts in which they are found. In form these morbid bodies are spherical, ovoid, or pyriform, their marginal contour in later stages becoming often crenulate. They are perfectly homogeneous in structure, devoid of concentric markings, colourless and pellucid, they may become slightly tinged by hæmatoxylin, but are wholly unaffected by carmine or aniline dyes, and they exhibit no reaction with the iodine and sulphuric acid test.

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[*] The following letter from Dr. Batty Tuke, inserted here by his wish, will serve to indicate how far he retains his former opinion with respect to the morbid change in question:—

"EDINBURGH, *May 24th*, 1889.

"DEAR DR. BEVAN LEWIS,

"After seeing your sections and comparing them with my own, I am convinced that most of the lesions described by Prof. Rutherford and myself many years ago are due, as you suggest, to myelitic changes. These changes are, for the most part, the result of the increase of the white substance of Schwann. I am gratified to find that you agree with what we said as to the material importance of these appearances in the morbid anatomy of chronic insanity. I am not prepared to give up the theory that certain of the changes we described may not be produced in other ways, and may not be due to degeneration of other brain elements. But, as I have already said, I am with you in the main.

"After your remarks on the subject it is unnecessary for me to enter upon any argument to prove that miliary sclerosis is not the result of the action of hardening agents. Such position is rendered quite untenable by the simple fact that the lesion is often demonstrable in fresh frozen specimens.—I am, yours sincerely,

"JOHN BATTY TUKE."

A case of **bulbar paralysis** occurring at the West Riding Asylum showed the lower half of the medulla to be the site of this lesion to such an extent, that its sections under a low power appeared as if besprinkled by thousands of minute droplets, and yet to the naked eye no abnormal appearance presented itself, and the section, although pale, was uniformly and fairly-well stained. A glance at the accompanying sketch (*Pl.* xvi.) will reveal the microscopic dimensions of these bodies and their wide-spread implications. Yet it will be equally obvious how absolutely the limits of the grey matter of the medulla is respected. Thus, in the olivary bodies we observe these morbid formations wholly confined to its medullated core, and nowhere implicating its plicated grey substance, *except where the latter is traversed*

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* "Lesions of the nervous tissues in the brain of the insane," *Brain*, Oct., 1879, p. 364.

by medullated fibres; and the same remark applies to the grey matter of the floor of the fourth ventricle, and nuclei of the cranial nerves.

The following is a *résumé* of the clinical features and pathological appearances in this case :—

T. W., aged thirty, married. He is a stone-mason, and was stated to have been insane for five months upon his admission. Two years previous to this date he was stated to have had a paralytic stroke. Five weeks prior to admission he again had a paralytic seizure (right hemiplegia), was deprived of speech, and became depressed and suicidal; great and increasing difficulty in deglutition had been noted since this second paralytic seizure. On admission to the asylum he was completely speechless, could only utter inarticulate sounds, or try to explain himself by gesture and pantomime. He appreciated all that was said to him, but showed considerable amnesia. When asked to write down his name he took up the pencil with his left hand first, and then, transferring it to his right, hesitated for some time as if trying to recall something, and then threw it down in despair. He expressed numbers by tapping successively with his finger on the table. He had been a steady man, of temperate habits.

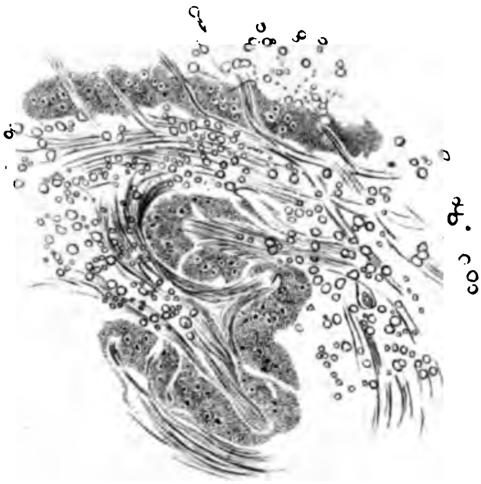
Circulatory, respiratory, and genito-urinary systems appeared normal.

His gait was somewhat unsteady, but there was no inclination to one side; the grip of the right hand is much diminished, and he uses his left hand in lieu of the right; no muscular wasting is apparent. The extremities are extremely cold, and both the feet and hands, as well as nose and cheeks, are livid; a similar patchy lividity is seen over the whole body. He fails to whistle or spit; cannot close his mouth, but opens it widely; saliva constantly dribbles from the mouth; he swallows fluid food only, and that with the greatest difficulty, throwing his head far back and accomplishing the act only after a prolonged effort, and then with much spluttering. The tongue appears completely paralysed, lies helplessly on the floor of the mouth, and cannot be protruded or laterally displaced.

Common sensibility and reflex activity appear normal and equal on both sides; perception of temperature and electric sensibility normal; all the affected muscles react energetically with feeble faradaic stimulation; all special senses appear normal. The pupils are dilated, the right pupil being the larger and more sluggish. For five years he remained a most anxious case for feeding, being in constant danger of choking. He had no further paralytic seizure, and died eventually of pulmonary gangrene.

Summary of Autopsy.—Bones of skull thickened and very dense; no adhesion of dura mater; the pia-arachnoid is opaque at the vertex, thickened, tough, and buoyed up by much serous fluid. In both hemispheres there is considerable atrophy of the convolutions, and where this wasting is extreme the cortex, after removal of the membranes, presents well-marked cauliflower puckering of the surface. The whole brain weighed about 900 grammes. The thickened membranes stripped with ease from all parts of the surface, except at certain sites where softening of the cortex had occurred. The softened patches were disposed with a certain degree of symmetry on both sides, thus :—

Right Hemisphere.	Left Hemisphere.
Slight along lower third of ascending frontal.	Lower half of ascending frontal.
Second annectant gyrus and cortex of inter-parietal sulcus.	Three upper annectants and boundaries of parieto-occipital sulcus.
Middle of third frontal gyrus.	



Portion of Inferior Olivary & Accessory Olivary bodies.
in a case of Bulbar Paralysis shewing
"Colloid Degeneration". × 22.

Bale & Dani

The patch of softening is generally of a greyish colour, translucent, and gelatiniform, its centre of bright yellow hue surrounded by a greyish translucent zone; it is pulpy and torn upon removal of the superjacent membranes.

The cauliflower puckering characterising the sites of most extreme wasting was disposed in the right hemisphere along the convolution bounding the longitudinal fissure, the middle of the second frontal and the angular gyrus; in the left hemisphere it involved the postero-parietal, the middle of the second frontal, and a portion of the second temporo-sphenoidal convolution.

Respecting the other organs of the body, the only point essential to note here (beyond the gangrenous condition of the lung) was the absence of any cardiac disease, and the presence of granular and wasted kidneys, somewhat extreme in both instances.

In the brain, also, we find these bodies encroach upon the grey matter only exceptionally, and then *invariably along the direction taken by the large medullated tracts* (the **tangential** fibres of the **peripheral zone** more especially), and more rarely the **intracortical arciform fibres** (*Pl.* xvii., *figs.* 1, 2). We have already alluded to the same limitation as regulating the distribution of the "**miliary**" patches.

No theory of the *connective origin* of these morbid formations could account for this peculiar restriction. Reverting to the case of bulbar paralysis (*Pl.* xvi.), we find these morbid products especially large and suitable for study along the fibres of the median raphé, the emergent root-fibres of the hypoglossal, and the arciform fibres near the raphé posteriorly. In these positions they lie either superimposed to the medullated fasciculi, or are crossed superficially by others; but, whenever their conformation assumes an elongate outline, their long diameter takes the direction of the medullated fasciculus lying parallel to the fibres. In many instances they are distinctly seen to be an oval swelling along the course of the medullated fibre, and the axis-cylinder can be traced through the centre of the swelling; in other instances a pyriform body presents itself, the narrow end of which is directly continuous with a swollen and deeply-stained axis-cylinder; or, again, a subglobose body, from the two poles of which the medullated axis is continued (although not traceable) within its structure. Some of the largest examples of the elliptic form attain the dimensions of 55 μ by 37 μ.

The presence of a nucleus within these bodies has been said to occur, giving, of course, much colour to the account of their cellular origin; with respect to this, we stated in the article already referred to, that such bodies were always extraneous. "Occasionally a nucleus appeared on the surface or border of these bodies, but it could always be regarded as extraneous to the morbid formation and accidentally superimposed."* Our methods of preparation now enable us to considerably extend such a statement; not only are they in all cases extraneous to the morbid swelling, but they are not *free* nuclei; they are really the nuclei of spider-cells (which are found when carefully looked for) attached to most of these so-called colloid bodies (*Pl.* xv.). So far from being, as we supposed, *accidentally* superimposed, they are important elements in the morbid *rôle*, their significance being identical with what has already been delineated in our description of "miliary sclerosis."

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* *Loc cit.*, p. 366.

The "colloid" body is, in its early stage, perfectly translucent and so minute that (unlike the miliary patch) it is not evident to the naked eye under reflected light (*Pl.* xviii. *B.*); it is likewise *attached*, forming an integral part of the medullated fibre, and, hence, not removable like the miliary deposit; it is, also, a single homogeneous body showing (of course) no stroma of fibrils through its structure; but these differences do not, we observe, indicate a distinct pathogenesis. Given certain conditions at a later stage, and the colloid bodies become opalescent or granular, swell to greater proportions, burst their albuminous sheath and coalesce as free miliary products, appear multilocular and have their structure permeated by the ramifying processes of scavenger spider-cells (*Pl.* xiv., *figs.* 2, 3).

The history is the same for all parts of the cerebro-spinal axis containing medullated fibres; but, as before stated, the transition stages are best studied where the larger medullated fibres exist in the region of the pons, medulla, and lateral columns of the cord. That this transition from the "colloid" to the "miliary" formations had occurred in the case quoted years since by Kesteven is, to our mind, conclusive. In his case the section of medulla of a patient, of whom the clinical history was unfortunately wanting, appeared under a low power to be full of minute cavities or perforations, which, when examined by higher powers, were found, in many instances, filled with a fine granular substance, similar to what we have already described as found in "miliary" patches. This drawing of the morbid groupings in the medulla reproduces the appearance met with in the case of bulbar paralysis already alluded to (*Pl.* xvi.), with this exception, that in the latter case the product of morbid activity was far more profusely scattered, and the individual bodies, of course, very minute (not having coalesced into miliary patches). Mr. Kesteven observes in reference to his case:—

"These cavities are irregular, scattered, without evidence of order, throughout the medulla oblongata referred to. They cannot be said to predominate specially in any one of the elements of the organ; but if they prevail at all in any part, it may perhaps be said that they are rather more numerous posteriorly than anteriorly. In one section, about the level of the calamus scriptorius, I counted several hundreds of these cavities." Again he adds:—"The surrounding textures appear to be perfectly healthy, with entire absence of any inflammatory action; neither is there sign of disease of the blood-vessels in the surrounding tissue. The morbid change is restricted to these detached points, and it is wholly a matter of conjecture whether it commenced in the capillaries, or in nerve-tubes or cells."*

Whenever this lesion appears in the spinal axis, it will be found advisable to study its nature in *longitudinal sections* and by the aid of *aniline dyes.*

To summarise our results, we regard both the "miliary" and "colloid" change as representing stages in the progress of a chronic degenerative affection of the medullated fibres of the centric nervous system; an affection which is of most frequent occurrence in the brain of the insane, and one of most vital import. A difference of opinion may exist regarding the special nature of the affection, whether it should be taken to indicate a simple degenerative change or one of chronic inflammatory irritation; and, in fact, the same question may be asked concerning the changes resulting from section of a peripheral nerve. In the one case, as in the other, the real origin of the affection is in the severance of the fibre from its trophic cell. It is in the diseased state of the cortical nerve-cells that we must seek, in most of our cases of insanity, for an explanation of this degeneration of the nerve-fibres; of course, any lesion causing severance betwixt the two, *at any site along the fibre*, will act in like manner, but the central disease in the cortical cell is usually the primary fact presented to us. The segmentation of myelin, occurring in this chronic affection, differs in some important particulars from what we see taking place in degeneration from section of peripheral nerves. The latter is apparently a more active process, and is the result of the direct morbid activity of the cellular constituents of the nerve-fibre; in its enlarged and dividing nucleus, and increased development of protoplasm, we recognise (as long since taught by Ranvier) the destructive agencies which bring about the segmentation and eventual destruction of the axis-cylinder and its medullary investment. In the more delicate fibres of the brain and spinal cord, segmentation of the myelin occurs more spontaneously; and as the medulla separates into varicose nodules along the length of the fibre, it becomes less susceptible to staining by Pal's process, which, in the healthy fibre, stains the medullated sheath of a deep purple, leaving the axis-cylinder untouched. It is then often noticed that the annular segments, although perfectly uncoloured for the greater part by this process, yet have a slight coloured fringe around both poles, the intervening medullated connection with the adjacent varicosity being normally stained and continuous with this coloured fringe. The appearance suggests an unchanged part of the medulla or its albuminous sheath at this site; possibly remains of the ruptured sheath. We must not regard varicosity of the fibres as conclusive of a commencing degenerative change; but when extreme varicosity of the larger medullated fibres is associated with their tendency to take up aniline and carmine staining, when they exhibit granular contents and clouding, and especially when apparently free granular masses with proliferating spider-cells are seen, we may be quite confident that we are dealing with a genuine degenerative change; finally, the presence of "colloid" bodies or of "miliary" patches assures us of the existence of the same condition.

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* See original article, "Notes of a Peculiar Form of 'Granular Degeneration' observed in a Medulla Oblongata." By W. B. Kesteven. *Brit. and For. Med. Chir. Rev.*, April, 1869.

Such extreme conditions of "colloid degeneration" (referred to by Dr. Batty Tuke as of occasional occurrence in the white substance of the brain, in which the section looks like "a slice of cold sago pudding") are undoubtedly states of degenerated medullary fibres from disease of their centric cells.

How far does this condition of the medullated fibre interfere with its normal conductibility or excitability? The long persistence of the axis-cylinder probably permits a free conduction along the fibre for some time after the latter is completely denuded of its myelin sheath; and we must regard this as still possible so long as actual severance of the axis be not affected. Such severance occurs (as we have seen) in the accidental accompaniment of "miliary sclerosis," and then sudden interruption must occur in the conductibility of the fibre; but, apart from such an occurrence the process is one of very chronic course, the denudation of the axis cylinder takes place very gradually, and the latter eventually succumbs to the encroachment of the sclerous tissue.

Granular Disintegration of Nerve-Cells.—The whole cell becomes swollen, and assumes a more spherical contour; the cell protoplasm loses its apparent homogeneity, and is clouded and obscured by the formation of granules within; the devitalised protoplasm no longer shows its affinity for the staining reagents, and becomes but faintly tinted by carmine or aniline dyes; the nucleus in like manner resisting these reagents. The nucleus often retires before the degenerating mass, is thrust aside, and becomes atrophied, shrunken, angular or elongated; moreover, the physiological pigment of the cell (usually found in a small collection at its base) becomes uniformly diffused, so that the altered granular protoplasm becomes of a yellowish tinge (*Pl.* xxiv., *fig.* 1). Meanwhile the lateral processes have become attenuated, and eventually dwindle down and wholly disappear; this gives the cell a still more globose aspect. In like manner, the apical process disintegrates, but the basal extension still remains, and is often notably swollen and prominent; it is seen in fresh specimens to be largely denuded of its investing medulla. These degenerate cells are mostly indistinct (from the absence of active staining), and some of the larger cells of the fifth layer in the motor cortex look like the ghosts of their former selves. Many of the smaller cells are found simply represented as a small heap of granules retaining more or less the outline of the original cell; the whole of the field around is the seat of much fatty granular matter, and especially accumulated around the blood-vessels. In the case of the latter we find the perivascular spaces greatly enlarged, the sheath enclosing fatty granules and deposits of hæmatine; the vessels are usually atheromatous, and fatty disintegrating branched corpuscles are spread on their exterior. The presence of much fatty matter is revealed by the fact that fresh preparations treated for a few seconds only by osmic acid (·25 per cent.) tend to become greatly obscured by a minute granular deposit forming over the surface of the section. The most accurate description of granular degeneration is, we think, that first recorded by Dr. H. C. Major, and certainly before his researches it had never been shown that a primary senile atrophy of the brain-cells occurred in senile dementia.* That observer also recorded similar changes in the cortex of aged animals, reproducing what he found in the human subject. Our further researches into the subject have resulted in the following observations. The early stage of granular disintegration of the cortical nerve-cells is signalised by certain remarkable features in the peripheral-zone of the cortex, immediately beneath the *pia mater*. Here the medullated fibres running parallel to the surface assume an extreme degree of varicosity, and active degenerative changes ensue. The elements of the lymph-connective system (spider-cells) proliferate and crowd around these varicose fibres, which now become moniliform from segmentation of the myelin, so that large globose or oval bodies unstained and connected by a narrow neck constituted by the stained axis-cylinder, are seen in large numbers beneath the *pia* (*Pl.* xvii., *fig.* 1). The bodies from being perfectly colourless and homogeneous, become clouded and slightly granular, and a dense proliferation of the spider-cells insinuates itself between and around these degenerating fibres, their branches forming a thick meshwork of fibres in this outer zone of the cortex. As in this stage the spider-cells stain intensely with aniline-black, we get in such specimens the contrast of numbers of colourless and somewhat lustrous spheres upon a dark background of felted fibre (*Pl.* xvii., *fig.* 1). This fibrous meshwork strikes down into the first cortical layer some distance beyond the limit of the medullated tract. This—the early—stage of granular degeneration is not so often seen, we much more frequently meet with the *next* stage, as in subjects dying from senile atrophy. It was this *early stage* of granular degeneration in senile atrophy that we drew attention to some twelve years ago, in an article on the lymphatic system of the brain;* we there sketched the appearances presented in such a section, and reproduce the sketch here, since it has been assumed by some that these features were peculiar to general paralysis (*Pl.* xxiii., *fig.* 1); we would here insist that *all* cases of senile cerebral atrophy exhibit this proliferation of spider-cells in the earlier stages of its evolution. Not only so, but we have already sufficiently indicated that we may expect to find similar appearances whenever these medullated fibres are degenerating, *whatever be the cause.*

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* *West Riding Asylum Reports*, vol. ii.

We see, therefore, reproduced in this layer of the cortex, in the fatty or granular degeneration of the nerve-cells, the so-called colloid degeneration already studied in the medulla and elsewhere. Do the same sclerotic results occur which we have traced in the latter? If this layer of the cortex be carefully examined (fresh sections) in the more advanced stage of this degenerative affection, we discover here and there a few colloid bodies remaining; but, in lieu of the long series of large moniliform fibres, or groups of large colloid bodies, and dense fibrous meshwork around, we find *free nuclei* undoubtedly arising from the spider-cells, scattered in numbers about, and each nucleus forming a centre, around which an abundance of highly refractile granules collect, which are of fatty nature (*Pl.* xxiii., *fig.* 2). These clusters of fatty granules around the free nucleus represent the disintegration of the spider-cell itself, for we often observe some of these elements full of glistening particles, and with their ramifying processes well seen lying amongst their disintegrated congeners.

The blood-vessels also at this stage have their sheath laden with fatty debris and refractile granules, like those surrounding the nuclei. It can be well appreciated how, under this process of fatty liquefaction and removal, this layer of the cortex becomes rapidly atrophied; the shrinking which occurs is apparent in the figure (*Pl.* xxiii., *figs.* 1, 2).

In considering the source of the fat we are met at the outset of our enquiry by Cohnheim's assertion that it is exceedingly improbable that lecithin could be converted in the organism into fat. Now, we are aware that nucleo-albumins, albumins, and lecithins (perhaps also nucleo-lecithins) are found in the cytoplasm, and the medullated sheath of nerve fibres; and if we find, as by Marchi's method, the degeneration of this proteid matter, and a replacement by fatty granules, we must infer that the latter either arose from the proteid, or are introduced *ab extra.* Miescher has shown that in the growth of the ova in Rhine salmon the wasting of muscular substance and the fat of the muscles, in other words, the *splitting up of so much proteid matter* issues in the large amount of nuclein and lecithin required for this

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* *Proc. Roy. Soc.*, No. 182, 1877.

Plate XVII.

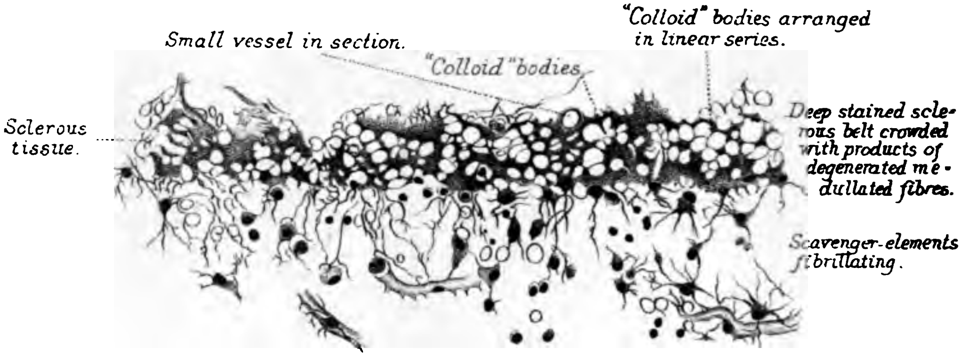
"Colloid" bodies arranged in linear series.
Small vessel in section.
"Colloid" bodies
Sclerous tissue.
Deep stained sclerous belt crowded with products of degenerated medullated fibres.
Scavenger-elements fibrillating.

Fig. 1.
"Colloid" degeneration of medullated arciform fibres in first layer of Cortex with active Scavenger cells.
Chronic Alcoholic Insanity. × 350.

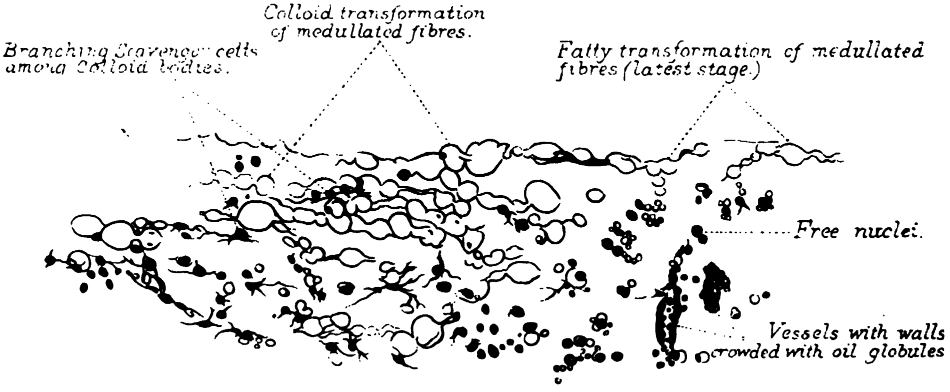
Colloid transformation of medullated fibres.
Branching Scavenger cells among Colloid bodies.
Fatty transformation of medullated fibres (latest stage.)
Free nuclei.
Vessels with walls crowded with oil globules

Fig. 2.
"Colloid" degeneration of medullated nerve fibre forming the arciform stripe in first layer of Cortex.
Advanced Senile Atrophy of Brain × 350

Bale & Danielsson, Ltd. Sculp.

physiological phase. Hence nuclein and lecithin do arise synthetically *within* the body by the splitting up of the proteid molecule.*

When, again, Hofmann showed that the eggs of *Musca vomitoria* could grow upon blood, and obtain all the requirements for the resulting grub from the proteid and cholestearin of the blood corpuscles, the same lesson was enforced. †

The immediate source of this fatty transfer may be referred to three categories:—

(*a*) Failure in the oxygen capacity of the nerve-cell; less oxygen is absorbed.

(*b*) Reduced oxygen value of the red blood corpuscles, such as we know to be the case in senility when the vital energies are at a low ebb.

(*c*) Interference with the blood supply—both from arterial degeneration and want of centric and circulatory energy.

We can only here presume that the cell nucleus plays a most important *rôle*, since the vital activity of the nuclear chromosomes appears to be the chief agency in the synthetic metabolism of the cell; and in the fact that the nucleus stains so faintly in these cases of senile degeneration, we see that a change has occurred in the chromatin which necessarily must be expressed in an altered nutrition of the cytoplasm. Instructive in this connection also is the fact that fatty degeneration ensues on the administration of arsenic, antimony, mineral acids, carbon monoxide and phosphorus, all of which restrict the supply of oxygen.

Pigmentary or Fuscous Degeneration.—The deposit of pigment in the nerve-cells of the grey matter of the brain and spinal cord is a constant feature in healthy states of these centres. So far from being in itself an indication of degeneration, its **absence** should at once make us suspicious of the integrity of the cell-unit, whilst its **presence** seems indicative, up to a certain point, of normal physiological activity. In some way, as yet not clearly understood, the presence of pigment plays an important *rôle* in the functional activity of the nerve-cell, and we need only refer to its abundance in the organs of special sense to emphasise this fact. We have seen in cases of granular degeneration, such as occurs in senile atrophy of the brain, that the earlier stages of decline in the functional vigour of the nerve-cell is associated with a *diminution* of its natural pigment. If the dementia has been ushered in by evidence of long-continued and great excitement, as in attacks of senile mania, then we find a notable degree of pigmentation of the degenerated cell far beyond what is seen in health.

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* See also evidence adduced by Mott in Allbutt's *System of Medicine*, Art. "Nutrition," vol. i., p. 188.

† Quoted by Bunge, *Physiol. Chem.*

Epileptic insanity and the insanity of general paralysis are, of all forms of mental ailment, those most prone to excessive pigmentation, but all morbid states of the nerve-centres which are associated with excessive and frequent engorgements of their vascular apparatus lead to the production of this increase of pigment; and thus, we find the same condition of the nerve-cell in certain cerebro-spinal tracts in severe chorea and even in so acute an affection as hydrophobia. To class this "fuscous" state with granular degeneration is we think misleading; the latter is truly a degeneration of the cell-protoplasm and may be associated, as we have just said, with increase or decrease of the normal pigment; the former is not truly a degeneration, but may be associated with an accompanying retrograde change in the neighbouring protoplasm, or, perhaps, may be its immediate cause. The one fact clearly established in the history of the various psychoses is that, where excessive pigmentation of nerve-cells is found, it is a witness to a bygone functional hyper-activity. Schafer* says:—"It seems to me that it (*i.e.*, pigment) is usually to be interpreted as a sign of activity rather than of decadence. For there is no doubt that in other organs the presence of pigment in cells is accompanied by marked protoplasmic activity, which may be both chemical and physical. I need only mention in this connection the hepatic cells, the pigment cells in the skin of the frog, and the hexagonal cells which form the outermost layers of the retina of the eye."

The large ganglionic cells of the cortex which are peculiarly prone to this excessive pigmentation offer us the best means for its illustration both in normal and abnormal states. In *fresh* preparations examined straight from the freezing-microtome, we find at one of the inferior angles, or along the basal arc of the cell, a small collection of golden yellow pigment, through which a number of dark, amorphous, minute granules are scattered; it appears to be surrounded on all sides by protoplasm, but is quite distinct from the latter; often it assumes a somewhat crescentic form partially encircling the nucleus. In degenerating cells, such as we have already referred to, the changes observed in the various stages are as follows:—First, the whole cell becomes tumid, and losing its more elliptic outline, approaches a somewhat pyriform or spheroidal contour, the pigment being notably increased in quantity. At the same time the cell-protoplasm stains of an intense depth of colour with aniline blue-black; so deeply tinged does it become that unless subjected to the dye for an unusually short period the whole of the unpigmented protoplasm and its contained nucleus becomes obscured (*Pl.* xxii.). With this intensity of staining of one portion of the cell we have the pigmented portion wholly unaffected by the aniline or carmine dyes, and assuming a bright yellow or brownish-yellow tinge, and a rough granular aspect. The cell becomes still more globose in aspect, and its numerous radiating lateral offshoots (at first coarse and deeply stained) can be traced *through the pigmented patch* up to the receding protoplasm. The nucleus is deeply stained by the usual reagents (*Pl.* xxii.). This appears to us to be the first stage of functional hyper-activity, and we find, as constant associates with these degenerating cells, coarse dilated blood-vessels, together with leucocytes and hæmatoidin crystals along the perivascular channels.

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* "The Nerve-cell Considered as the Basis of Neurology," *Brain*, 1893, p. 136.

The retraction of the unaffected protoplasm carries with it the nucleus towards the apex of the cell, or draws it out eccentrically and to the side; but at times the invasion of the pigmentary change appears at the summit of the cell when the nucleus and investing protoplasm retreat towards the base. The nucleus itself may now become pigmented in some cases, but in all it assumes eventually a more or less irregular angular contour, losing the plump, oval contour seen in fresh and healthy sections. At this juncture, also, the staining of the cell by aniline becomes less evident, and, with the encroachment of the ever-increasing pigmented area, faintly stained tracts or angles of protoplasm may alone remain. The radiating lateral processes dwindle down into extremely attenuated extensions and entirely disappear, the cell being devoid of all except a basal and perhaps a short apical stump; in others, a few bristle-like projections from the sides of the cell still remain, so that it has a somewhat spiny aspect. In this stage, whatever processes remain are but very faintly stained by reagents, or have a granular, degenerating aspect, while the pigment discoloration can often be traced far down the process from its junction with the cell. The pigmented area appears to be separated from the remaining protoplasm of the cell by an investing capsule of more deeply-stained material, so that when the greater part of the cell is involved in the change, the latter appears to possess a very definite investing-wall, deeply-stained by aniline, with brownish-yellow, granular pigment within (*Pl.* xxi.). Such a sharply-defined, indurated border gives the cell the appearance which has been described by Meynert and Lubimoff as a "sclerosed swelling." At this period the nucleus, besides presenting an irregular contour, exhibits one or more highly-refractile spots, probably of fatty nature; it remains always the centre around which any non-pigmented and unaffected protoplasm, which is left, collects. Hence, many cells in an advanced stage of degeneration exhibit an eccentric nucleus surrounded by a narrow zone of stained (and, hence, presumably still healthy) protoplasm, delicate extensions of which can be traced as dark fibres running through the investing pigmented granular cell-mass, the whole being enclosed within an irregular, distorted, dark-stained sclerous envelope. We have also often observed a sharply-defined cincture separating the healthier from the degenerate portion of the cell; and it may always be noted that the processes which arise from the pigmented area are more degenerated than those issuing from the healthier stained segment. The individual granules seen in the pigmented mass are from 1 μ to 2 μ in diameter.

The last stage is that of general shrinking of the cell, which is, however, preceded by a partial resolution of the bright yellow or dusky pigmented granules into many highly-refractile globules, more obviously fatty in nature; whilst this admixture diminishes the fuscous aspect of the cell (*Pl.* xxiv., *fig.* 1; *Pl.* xxvi.). In many, a still more complete transformation is apparent; the yellow tint wholly goes, the cell is filled by a somewhat bright, translucent colourless material, finely granular or molecular in part, and the outline of the cell is so faintly mapped-out that it may be easily overlooked. These shrunken cells are also found broken up into little heaps of colourless or faintly-pigmented disintegrated molecules (*Pl.* xxiii., *fig.* 3).

The behaviour of the chromophil granules has been noted and admirably figured by Meyer. In the earlier and intermediate stages these granules, greatly reduced in size, give to the swollen cell a finely-punctated aspect, the dissolution of the granules giving place to pigment.*

The granular pigment in the fresh sections is apparently unaffected by ether, by alcohol, or by both conjointly; nor does it undergo any obvious change with caustic soda or fuming nitric acid. All such pigmented collections, in cells advanced in degeneration, show a decided darkening when treated with osmic acid (1 per cent.), and thus reveal a certain proportion of fatty constituents; whereas the pale cell, full of translucent material, shows decidedly a fatty reaction when so treated. We have already noted that the pigmented portion takes up none of the usual dyes—carmine, hæmatoxylin, or aniline. To summarise briefly the changes thus undergone by the cell, we may arrange them under three periods, thus:—

Period of over activity.—	(1) Swelling of cell with increase of pigment.
	Dark staining of protoplasm, nucleus, and branches.
	(2) Advancing degeneration, cell more globose; protoplasm retracting.
	Sclerosic investment of cell and cinture formed.
Period of diminished activity.—	Nucleus eccentric, deformed, fatty, with narrow encircling zone of protoplasm.
	Processes few; these, as well as cell-protoplasm, faintly stained.
Period of absorption.—	Fatty transformation and decoloration of cell.
	Atrophy with shrinking or rupture into a heap of granules.

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* "Types of Changes in the Giant Cells of Paracentral Lobule," by Adolph Meyer, *American Journ. of Insanity*, vol. liv., No. 2.

During the progress of the fatty transformation vacuolation of the cell not infrequently occurs; and it is from this cause that it appears occasionally full of loculi, the fatty contents of which seem to have dropped out or to have been absorbed, the walls or dissepiments of the several loculi remaining rigid. Such cells present a very extraordinary appearance (*Pl.* xix., xxi.), and we are ignorant as to the cause which induces this transformation rather than the more usual fuscous change and atrophy.

Developmental Arrest of the Nerve-Cell.—At an early phase of its history the cortical nerve-cell of the human subject is of spheroidal contour, its basal process non-medullated, and the cell itself possessed of extremely few processes. Not only so, but the cells are of small size, and are much more uniform in their dimensions throughout the depth of the cortex than in the fully-developed and adult brain. So utterly unlike are these young cells to the form ultimately assumed in the fully-developed stage, that the one cannot possibly be mistaken for the other. When, therefore, this type of cell prevails at a later period of life, we have unanswerable testimony to its arrested development. It might, of course, be objected that such primitive cell-forms, occurring in youth and adult life, were products of a retrogressive process and not absolute proof of their arrested development; and this view might be supposed to be strengthened by the fact that in the granular and granulo-pigmentary degenerations already described we have reproduced inflated cell-forms, which at a certain stage possess characters strongly reminding us of the primitive cell.

Such a conclusion is, however, inadmissible, since these degenerative processes are progressive, and result in the complete disintegration of the nervous elements; since we find in these cases cells side by side in every stage of degenerative change; and since, in the great majority of the cells involved, special features present themselves which are not afforded by the cells of stunted cerebral development to which we now allude.

It appears to us that too little attention has been bestowed upon this important evidence of developmental arrest; we drew attention to its occurrence in certain instances of epileptic idiocy and imbecility in the year 1879,* and since this period we have had frequent opportunity of verifying the observations then made.

The condition to which we allude we find restricted to the **convulsive neuroses**; hitherto we have failed to note its presence in **simple forms** of **congenital defect** and deaf-mutism; all the instances falling under our notice being subjects of **epileptic idiocy.**

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* *Brain*, Oct., 1879, p. 371.

It is important to note, in the first place, that the cortical layers presenting this primitive type are especially the second and the third layers; the larger ganglionic cells are usually in a state of excessive pigmentation, and even present evidence of the granular degeneration such as we usually meet with in epileptic insanity; but, apart from this, they do not assume the characters presented by the elements of the superimposed layers, and they usually retain their normal outline.

Our first glance at the cortex in the second and third layers, through a low-power objective, suggests to the mind a staining of the nuclear elements only, the faint delineation of the cell escaping attention; but it soon becomes obvious on more careful search that the nerve-cells are there in apparently undiminished numbers, but the majority *completely unaffected* by the staining reagent employed. Their appearance is made obvious, in fact, by the presence of pigmented (or else colourless, but translucent and often highly-refractile) contents which *completely fill the cell.*

We have alluded to these cells as spheroidal, and in many instances such is the case; but, perhaps, the slightly-pyriform contour predominates. They resemble, in fact, a number of delicate, yellow, pear-shaped bladders suspended by a stalk—the stalk being the faintly-stained apex process, whilst, at the junction of the latter with the cell, the well-stained nucleus presents itself. The only elements which stain normally with the aniline dye are the large cells of the fifth layer, and these (as before stated) are often degenerated.

With greater amplification the cell-contents are found to be granular throughout—not as in the degenerative change previously described, such as exists in senile atrophy, limited to one portion of the cell, and contrasting strongly with the stained protoplasm elsewhere, but *uniformly*, and coarsely granular, resolved by high powers into oval or spherical bodies usually 2 μ to 4 μ in diameter. Such granules are often highly refractile and quite colourless, but usually are pigmented of a bright yellow tint. Certain cells exhibit a faint staining between these granular bodies, giving them a somewhat reticulated aspect; this undoubtedly indicates the existence of the original protoplasmic groundwork of the cell unaffected by pigmentary or fatty change, and, in some instances, a somewhat dark-stained border surrounds the cell, forming a well-defined outline—this is, however, exceptional.

The eccentricity of the nucleus is a notable feature, its usual position being at the junction of the apex-process with the cell, but it is occasionally appressed and flattened against the sides of the cell; it is of fair proportionate size in most cells, is deeply stained by aniline, and often presents one or more refractile spots in its interior. The branches radiating from these cells always stain *very feebly*, are very delicate and attenuated, and the paucity of branches is one of the most notable features of the cell; in the greater number of instances the apex-process is alone detected. Most cells show only two divergent branches near the inferior pole, whilst it is rare to meet with four or five processes. We may thus summarise the features presented by these nerve-cells of the upper layers of the cortex:—

(*a*) Spheroidal or pyriform contour of cells.

(*b*) Marked eccentricity of nucleus, usually apical in position.

(*c*) Coarse granular condition of contents.

(*d*) Pigmentation universal, or indications of a fatty change of protoplasm.

(*e*) Great paucity of branches.

(*f*) Peculiar characters far most marked in cells of second and third layers.

We have, therefore, in the upper cellular zones of the cortex in these cases of mental defect associated with epilepsy:—

1. The **primitive type** of cell reproduced as regards contour and branching;

2. But stamped of a **degenerate type** by the granulo-pigmentary or fatty condition of its contents.

It would seem to us that the latter condition is not an active degeneration, but rather the *natural state* of a degenerate type of cell, as it does not proceed to the rapid disintegration of cell-structure which pertains to the ordinary granular and "fuscous" degenerations of later life; and, as we have seen, it is not at any time found as a partial, but as a universal, condition of the cell-structure. In epileptic insanity where the epilepsy is acquired at puberty or at adult age, however frequent and severe the convulsive seizures, however long-standing such phenomena have been, we never find reproduced the appearances above detailed. However advanced the "fuscous" or granular change, we find the degenerating cells, if once they have acquired their normal developmental characters, show indications of the mature type to the very end; and, hence, we can in no way consider the very peculiar conformation of these cells in epileptic idiocy to be the outcome of a primary degeneration. We shall again allude to these stunted globose cells when dealing with epileptic insanity.

Vacuolation of Nerve-Cells.—This change consists in the appearance within the nerve-cell of oval or perfectly-spheroidal bodies, of high refractile power quite unaffected by any staining reagent, colourless but lustrous. In many cases the lustrous, refractile quality may be wanting, and it is then evident that the spheroidal outline is that of a genuine *cavity* or *vacuole*, from which the former contents have been removed, or escaped by rupture; that such rupture of the cell does occur is sufficiently evident in the case of certain elements where an incomplete vacuole is apparent along the border of the nerve-cell, which is interrupted here by a wide opening leading into the cavity of the vacuole. There may be but one such vacuole formed in the cell-protoplasm, but we frequently find many such within each cell; and, in extreme cases, they crowd the interior so as to present the very remarkable appearance indicated in the accompanying figure, where the outlines of *eighteen vacuoles* were seen in a single large multipolar cell. The removal of the contents of such vacuoles may be effected by reagents, by the methods of preparation of the section, and, as we believe, by direct absorption during life through the agency of the **lymph-connective system.** However removed, it is evident that the original cavity maintains its former contour, and is never encroached upon by the protoplasm surrounding it; and in such cases where the cavity has opened up on the outer surface of the cell no retraction of the protoplasm occurs, but the contour is rigidly preserved. The protoplasm surrounding the vacuoles is more or less in a state of granular degeneration, faintly stained, or pigmented and fuscous. This association of vacuolation and granular degeneration is invariable (*Pl.* xxi.); yet the vacuoles are often *immediately* in contact with unaffected protoplasm which assumes a deep-stained tint, and still further aids in bringing them into relief. The nucleus of the cell may be concealed from view or really absent; usually it is considerably displaced. The aspect of many of these degenerated cells is suggestive of **encapsulation,** through the formation of an outer delicate investing pellicle of devitalised protoplasm (see lower three cells in *Pl.* xxi.). The granular degeneration and the resulting vacuolation and feeble staining of nerve-cells indicate a fatty change in the cell-protoplasm, and the separation of the fatty matter which fills the vacuole can, as is now well known, be artificially induced; thus in *phosphorus poisoning* we are aware that an acute fatty degeneration occurs in the tissues from an increased metamorphosis of albumen, but chiefly from interference with the oxidation of the tissues, and, hence, the accumulation of fat within the cell. This is mainly due to the destruction of the oxygen-carriers—the red blood-corpuscles—induced by phosphorus* (*Voit and Bauer*). This rapid splitting-up of the protoplasm of the cell, and the accumulation of fat within its structure, is equally induced in the *nerve-cells*; and the experiments of Voit and Bauer have been repeated upon dogs and rabbits by Popow, Danilo, Kreyssig, and Flesch, with results which indicate that phosphorus and arsenic apparently induce a granular degeneration and vacuolation of the ganglion cells of the spinal cord. Flesch, however, and, later on, Trzebinski, have questioned the conclusions arrived at by other authorities, and would assign the vacuolation to an alteration induced by chrome reagents. The latter emphatically asserts that in fresh preparations examined by him the change was never witnessed.* We have on the contrary not only constantly met with vacuolated cells in fresh frozen-sections of certain subjects of insanity, but the most extensive instance of this degenerative change we have seen, and which we have figured in *Plate* xxi., was treated entirely by fresh methods (sections from frozen brain being stained with aniline blue-black). We are, therefore, assured that Flesch is certainly premature in the conclusion arrived at, viz., that vacuolation is not met with in nervous tissues examined fresh, but is presumably induced by hardening reagents. Trzebinski's researches indicate that these changes may be imitated by the use of chrome reagents in *healthy* tissues; but they do not prove the artificial nature of these changes in *diseased* nervous centres, since we repeatedly meet with these vacuolated cells in our *frozen-sections* of brain.†

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* *Zeitschrift für Biologie*, vii., *Voit and Bauer*.

The vacuolation is, as before stated, always associated with the granular degeneration, although the latter may frequently be found to afford no instances of vacuolation.‡ We meet, therefore, with this change in the cell in senile cerebral atrophy, and it is by no means an infrequent condition in the insanity of chronic alcoholism. In both cases we must attribute it to the accumulation of hydrocarbon in the tissues from defective oxidation, which is the invariable accompaniment of old age, and to excessive indulgence in alcohol.

Vacuolation of Nucleus.—The former condition is usually confined to the nerve-cell, the nucleus not necessarily being implicated in like manner; and it is a feature more common in the *large* ganglionic cells of the spinal cord or the motor area of the cortex of the brain, than in the cells of the posterior cornua, or of the superjacent layers of the cortex; at all times they are more prominent and obtrusive appearances in the former positions.

The nucleus-change, however, is one peculiarly common to the smaller cells of the upper layers of the cortex; and, in fact, is often limited to the second layer—the small angular cells, fringing externally the small pyramidal elements of the third layer. It is exceptional to find so notable and so extensive a change as that represented in *Pl.* xx. On referring to that drawing, we observe that almost every cell is vacuolated; some containing several vacuoles, and most presenting unnatural, distorted outlines as the result of the change undergone. It will also be observed that the **nucleus** is, in the univacuolated cells, the **primary seat** of disease; so that in many cases the whole nucleus is represented by a spherical vacuole, and the cell, in lieu of its dark-stained centre, shows an unstained bright spheroid surrounded by the stained protoplasm of the cell. Early indications of this change are revealed by a minute oily droplet in the centre of the nucleus; such droplets multiply and progressively enlarge, until, no longer coalescing in the nucleus, they become free within the cell-protoplasm, which is also found in a state of granular degeneration (*Pl.* xx.). The change as peculiarly confined to the nucleus in early stages, will be more carefully considered in our section on the pathology of the insanity of epilepsy, since it is in this and certain other convulsive affections that we meet with it as a very notable and uniform change.

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* *Arch. für Path. Anat. u. Physiol u. für Klin. Med.*, Bd. cvii., H.I. or an abstract of the paper by *Dr. Ernest Birt* in *Brain.*

† See "Remarks on a case of phosphorus poisoning, with special reference to the mental symptoms during life, and the pathological appearances in the Brain Cortex after death," by F. A. Elkins and J. Middlemass, *Brit. Med. Journ.*, Dec., 1891; also "Vacuolation and Phosphorus Poisoning," by Dr. Macpherson, *Lancet*, May, 1892.

‡ "Vacuolation of Nerve Cells and Septic Infection," *Journ. of Pathology*, Feb., 1894.

It may at first appear an unnecessary refinement to distinguish between the vacuolation of cell and of nucleus as we have here done; both are indications of a fatty change finally producing the self-same disintegration. It is, however, important that such a distinction be drawn, since the site of lesion in both instances appears to us to indicate a wholly dissimilar origin. The fatty disintegration and vacuolation of the large ganglionic cells appear to be induced by changes in the blood-corpuscles leading to defective oxygenation, by chronic pulmonary affections acting in the same way, by the effects of certain poisons (arsenic, phosphorus) or any of the many circumstances which restrict the supply of oxygen to the tissues; the effect is a general one, but those elements in the cortex naturally suffer earliest and most severely whose nutrition is carried on at greatest disadvantage. This is peculiarly the case with all the large-sized cells of the cortex, whose bulk and greater distance from the arterial twigs is inimical to rapid restoration of nutritive equilibrium, as long since indicated by Dr. Ross.* These large cells, therefore, are the first to succumb to fatty change induced by any general effect restricting oxidation.

When, however, we meet with a special layer of the cortex, and more especially of its smallest nerve-elements so affected, the same explanation is not valid; we cannot imagine these minute elements suffering so extensively, whilst the larger escape from any widespread defect in oxygenation. We can only here presume that the change induced is indicative of an *intrinsic morbid factor in the cell itself*, or of its immediate structural connections. Hence we regard the changes found in senile atrophy of the brain-cell as having an entirely different significance to those found in epilepsy and in chronic alcoholic insanity; in the latter we do not look for a cause of the

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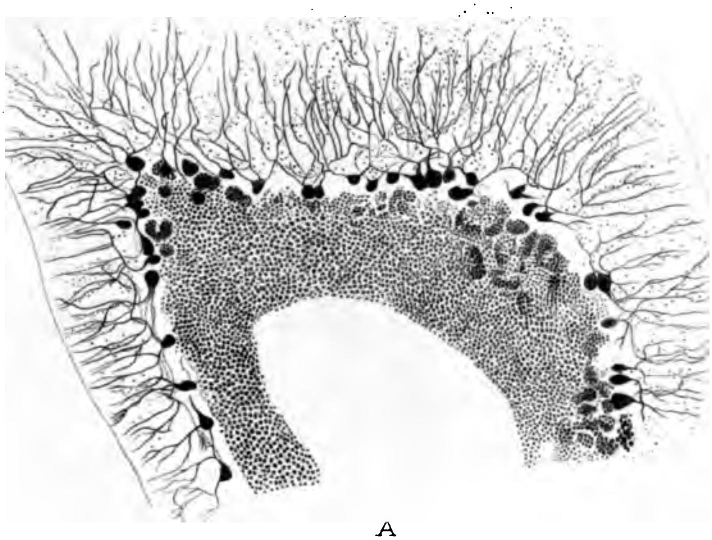
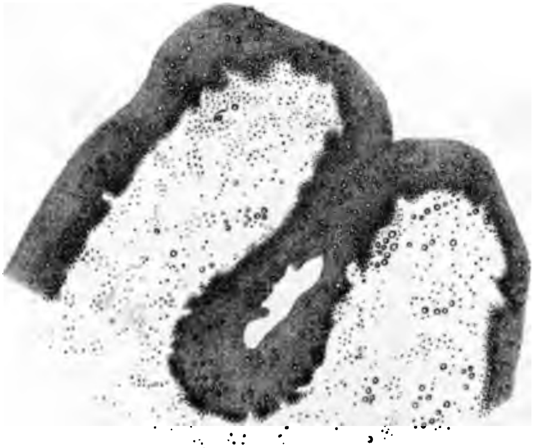
* *Diseases of the Nervous System*, vol. i.



A



B.

Sclerosic implication of Cerebellar Cortex.
in a case of Epilepsy.

A. Healthy leaflet closely adjoining diseased tract.
B. Sclerosed leaflets united firmly together.

Bale & Danielsson

degenerative change in the blood or vascular apparatus, but in some primary condition of the nervous arrangements in themselves.*

Destruction of Nerve-Fibre Plexus.—In cases of chronic insanity, and especially where atrophy is a prominent feature in the cerebral hemispheres, the fresh cortex obtained by freezing, and stained by aniline blue-black, shows highly characteristic appearances. In *healthy* brain a clear differentiation of the nervous elements, the cell and nerve-fibre plexus, prevails, when fresh sections are so treated; they appear darkly-stained, and imbedded in a clear, unstained matrix, in which connective nuclei and meandering vessels are less obviously defined. In the chronic atrophic diseases of the cortex associated with insanity the aspect is very different. Here we find much **diffusiveness** of staining, the matrix being uniformly affected by the aniline, or exhibiting a patchy mottling of a deep aniline tint separated by perfectly light or faintly-tinted areas.

In consequence of this diffuse staining of the matrix, which often approaches the depth of tint assumed by the nerve-cell, the differentiation of the latter is greatly impaired; and we have known these specimens thrown aside as badly-prepared sections, the blurred and indistinct elements being regarded as failures in staining rather than the results of morbid change. In all advanced cases the uniform diffusiveness of staining is much intensified by the degenerative changes proceeding in the nerve-cell and its network of branches; but, it is important to note that, the former long survives the latter as an essential though diseased element in the cortex. It is the fibre-plexus formed by the radiating processes (not the primary but the secondary) which are earliest implicated, and we frequently note their almost entire absence from the field, whilst the degenerate cell remains in various stages of decay.

In cases of secondary dementia, we always note the appearances now detailed, especially in the anterior sections of the hemispheres; there is the great paucity of cell-processes, the patchy mottling of the intercellular areas of the matrix, an increase in nuclei, and the field strewn with the faintly-stained, indefinite, blurred outline of degenerating nerve-cells. The patchy mottling, on closer examination, resolves itself into the fine fibrillar meshwork which originates from the scavenger-elements (spider-cells), and which has replaced the non-medullated meshwork intervening between the nerve-cells: but partly into coarser patches resulting from the disintegrated nerve-cells, which have entirely lost all semblance of their former outline, or being well-defined in their lower half, become indistinct above, and fade off imperceptibly into the surrounding matrix; or they may have attached to them a few shrunken nuclei as sole representatives of the spider-cells which attacked them at an earlier stage.

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* Consult on this point the following recent articles :—" Vacuolation of nerve-cell nuclei in the cortex in two cases of cerebral concussion," by J. Macpherson, M.D., *Lancet*, May, 1892; " Vacuolation of nuclei in Myxœdema," Dr. Whitwell, *Brit. Med. Journ.*, vol. i., 1892; " Vacuolation of nerve-cell nuclei," by Dr. Skae, *Brit. Med. Journ.*, May, 1894, and by Dr. Wynne, *Brit. Med. Journ.*, June, 1894.

The fact last alluded to is important. It must be borne in mind that these destructive elements have but a transient period of existence, and the more actively they play the part of scavengers on the neighbouring tissue, the more rapidly (we may assume) do they fibrillate and lose their cellular constitution, passing in this stage through a fatty transformation like the nerve-cell (*Pl.* xxii., *fig.* 2).

In advanced instances of this interstitial atrophy, we consequently may find few, if any, of these characteristic organisms present themselves; but the resulting fibrillar meshwork is always perceptible. The fatty change of these tissue-elements involves considerable discoloration by the use of osmic acid, which is requisite in the fresh process of examination, and it therefore becomes imperative that a very dilute solution of the reagent be employed, unless the firmness of tissue permits of its being entirely dispensed with; if the usual ·5 per cent. solution be employed, great obscuration of the tissue-elements may result.

The delicate, unprotected processes of the intercellular nerve-plexus lend themselves most readily to the ravages of the scavenger-cells; they succumb sooner than the apex-process, the cell itself, and the basal axis-cylinder process, which persist latest (*Pl.* xxiii., *fig.* 1). Hence, as we shall find in most instances of secondary dementia, it is this plexus which is earliest and most exposed to decay, and from it issues that interstitial atrophy which progressively advances to the termination of the case; it is a destruction of tissue which can never be replaced.

A due estimate of the nerve-cell as the elementary unit of the nervous mechanism is now universally recognised as essential to an intelligent appreciation of the phenomena of cerebral activity, whether from the physiological or pathological standpoint. That the absence of the elaborate cell-mechanisms of the cortex, and their imagined replacement by a perfectly homogeneous structureless matrix in which the nerve-fibres lost themselves, would introduce into our problems in the present state of physiological science, inextricable confusion, is self-evident; since the phenomena of functional differentiation would then remain to us a profound mystery, and the simplest correlations of mind would await an explanation.

If for these reasons only, we may safely exalt the nerve-cell to a position of the very highest importance in our problems of mind. It is on this account highly important that the nerve-cell of the cerebral cortex should be the subject of careful study by all interested in psychological medicine; and that due attention should be paid to the conditioning of its functional activities, and to the results of its nutritional impairment, disease and death.

In our chapter on the histology of the cortex, we have dwelt sufficiently upon this structural unit in its relationships to the surrounding elements, to indicate the intricacy and delicate adjustment established betwixt it and its physiological environment. Suspended within a sac in direct connection with the lymph channels surrounding the blood-vessels—or rather its own special nutrient capillary—the nerve-cell becomes liable to any influence, however trivial, disturbing the blood-stream in its neighbourhood (*Pl.* xii.). A quickened circulation, a retarded flow, an anæmic state of cortex must influence the functional activity of these centres of feeling and thought; a vitiated quality of the blood, or the presence of toxic agents introduced from without, or elaborated within, the economy will all affect them in a greater or less degree; whilst the activity of the lymph-connective system in the removal of the effete products of functional wear and tear, will play an all-important *rôle* in the same direction.

That delicate system of **lymph-connective elements,** to which we have alluded as permeating (in the normal state) the whole of the cerebral mass of white and grey substance, takes a more active share in the pathogenesis of mental decadence than any other: and the more the question is investigated, the greater importance, we feel convinced, will be attached to these elements in the processes of disease as affecting the nervous centres. Their **physiological** indications are clear; they are the **scavengers of the brain;** and the evidence obtainable renders it now incontrovertible that they are liable to excessive and rapid development under certain morbid conditions affecting cerebral nutrition and repair. In the normal condition of healthy cerebration these elements, far from being obtrusively present, are so delicate and pellucid that they often escape detection; but that they are universally present can be readily verified by special methods of examination.

Whatever leads to increased **waste** of cerebral neurine; whenever **structural disintegration** is slowly proceeding either in nerve-cell or fibre; whenever **accumulation** of **debris** occurs from disease of the vascular tracts, then we invariably note an augmented activity registered in these **scavenger-elements** of the brain. That their activity is in direct ratio to the functional activity of the essential neurine tissue, we think there can be no doubt; nor that with each accession of the nerve-tide they are stimulated to increased activity in the removal of the products of waste and the plasma effused from the vessels. In *healthy states*, however, they never assume the hypertrophied form, the deep staining, the coarse fibrillation, the rapid multiplication, and the evidence of obvious intracellular digestion, which are so readily observed in pathological states (*Pl.* xxv.).

Tissue-Degradation from Over-strain.—In certain pathological states, notably in general paralysis, we shall find that these organisms play an all-important part, and are most prominent factors in the morbid process; but we desire here to draw attention to their connection with certain states on the very borderland of pathology, viz., instances of "over-strain," where cerebral activity has been too long or too intensely encouraged, and mental tension has been associated with worrying and distracting circumstances. In such cases, as all of us are aware, there is a risk of **permanent damage,** and most of us are acquainted with instances of such. That sustained mental work indulged in by the healthiest subjects, yet with disregard to physiological laws, will reach (if persisted in) a limit where the pathological barrier is passed, is undoubtedly true; but that the introduction of the element of **worry,** interfering with the smooth current of intellectual work, *has a specially vicious influence* in this direction is a fact of such far-reaching consequences, that it cannot be too forcibly or too often insisted upon. No amount of rest from mental labour, no change of circumstances, nor absence of all disturbing agencies will, in the cases we refer to, restore the mental faculties to their former vigour; it would seem that an actual **destruction of tissue** has occurred as completely as if excised by the scalpel, and that restitution to the former state is impossible. We believe that in such instances an actual degeneration of cerebral-tissue has been entailed, that the lymph-connective system has received just such a stimulus beyond the physiological limits necessary to ordinary repair, that these physiological units become pathological factors, and the nervous elements themselves succumb to their rapacity.

Tissue-Degradation from Active Pathological Processes.—In **consecutive dementia** following upon acute insanity, we have evidence that what we have portrayed above occurs to a very wide extent in the cortex of the cerebrum. That it occurs in a minor degree in *all attacks* of mania and acute melancholia is also very probable, judging from the almost invariable signs of instability, and slight enfeeblement of potential nerve-energy in most apparent recoveries; but for consecutive dementia the actual fact stands out as a prominent and highly-significant feature, that there is *very obvious* destruction of the nerve-fibre plexus of the cortex, and that the intercellular elements have degraded in type to the purely connective, or have undergone fatty disintegration and removal.

We regard the appearances presented by such cases (already described, p. 537) as indications of the storm which overswept the region, and as evidence of repeated engorgement of the cortical vessels attendant upon the hyper-activity of the nerve-elements. We see in such instances the coarse and tortuous blood-vessels, the frequent minute extravasations and deposits of hæmatoidine in their neighbourhood, and other changes incident to bygone attacks of excitement; but in the presence of the spider-cells and the degraded type of issue, we see an actively-destructive agency at work, which has, therefore, a very different significance to the vascular changes associated therewith.

In no case of acute *uncomplicated* mania or melancholia, fatal in early days, have we met with these organisms as pathological factors; it would seem that the normal elements play their part as tissue-scavengers, with a fair promise of success up to a certain limit of time. In fact, the **duration** of the **excitement** is of primary importance in the elaboration of these morbid factors; this we shall have occasion to see again and again in our clinical studies of insanity, where the **time-element** is of such great moment that it is customary to assume (arbitrarily of course, for many exceptions occur) that cases of mania of over twelve months' duration may be relegated to the chronic class of the insane. There is, indeed, but little doubt that beyond a certain period, varying for each individual's neurotic resistance (whether it be within or beyond twelve months' duration of mental excitement), these elements multiply and take up a pathological *rôle*, preying upon the tissues whose functional integrity in health they subserve.

Another factor should, however, be taken into account in this connection—viz., **Age.** The tendency in advancing age is towards a multiplication or overgrowth of these elements, so that in senile dementia (as we shall see) they form the natural associates of the atrophic changes which accompany the decadence of mind. This tendency is, therefore, emphasised in all acute insanities occurring in advancing years; and we may regard *age* as an important element in determining the consecutive dementia resulting from all forms of insanity.

Tissue-Degradation from Disuse.—The process we have been considering is one of genuine degradation of tissue preceded by over-stimulation of cortical areas; but, a degradation in type may also be slowly induced (according to well-known physiological principles) by lowered functional activity or **disuse.** The atrophic changes then resultant are induced through the agency of the same scavenger-elements which remove the degenerating tissues and replace them by their own fibrillated stroma. We are thus inclined to explain the profusion of these spider-elements in the cortex of most domesticated animals, notably in the sheep and in the tame rabbit. In the former, the pia is firmly attached to the surface of the brain by these elements, and requires considerable force for its removal; in the latter, sections through the cortex exhibit them in great abundance in the peripheral zone. Similarly they are to be noted in the cat, dog, ox, and monkey in varying degrees. Sir J. Crichton-Browne has called attention to the disparity in weight between the brain of certain wild and domesticated animals, taking as a forcible illustration the reduced weight of the brain of the tame, domesticated duck as contrasted with that of its wild representative. The decadence from disuse of certain faculties essential in the wild state is thus actually expressed in weight, and we might infer that in all alike a degradation of tissue initiates such atrophic changes. In man a similar change suggests itself as occurring in those instances where a long life of unusual mental and bodily activity has been suddenly interrupted by circumstances which restrict within an extremely limited range his further activities; in such instances, how frequently do we date the first evidence of mental decadence from the cessation of the customary active life of the individual. The preference to "die in harness, rather than rust" is a trite saying, dictated by a full appreciation of the physiological principle of the decline of faculties with their disuse.

Since then, these lymph-connective elements play so important a *rôle* in the degenerations of cerebral tissues in mental disease, it becomes an interesting question to enquire how far their pathological development may reveal itself to the naked-eye examination of the morbid brain? The presence of unnatural attachments betwixt the pia-arachnoid and subjacent cortex as morbid adhesion is conclusive evidence of the presence of these pathological elements; even undue firmness of connection, apart from actual adhesion, may indicate their presence (as we have seen, occurs in the brain of the lower animals), but their presence in the cortex already softened by an acute process, such as occurs in general paralysis of the insane, is accompanied by the most pronounced appearances of morbid connection.

Yet, although their presence is confirmed by the morbid adhesions so affected, it by no means follows that the *absence* of adhesion necessarily excludes them from this site; in fact, these elements in a state of morbid proliferation in the outer zone of the cortex may be frequently seen where no suspicion of adherent membranes exists (*Pl.* xxiii., *fig.* 2). In the latter case, their appearance is usually coincident with considerable **fatty disintegration** of tissue, these elements themselves succumbing to this change, and very considerable interstitial atrophy is apparent.

Another indication of the ravages of these scavenger-cells is afforded by the various *atrophic states* presented by the cortex of the chronic insane —of course, exclusive of such atrophy as is dependent upon obviously gross lesions—apoplectic foci, softened tracts, &c. (*Pl.* xxiii., *fig.* 1). Such atrophy is always attendant upon the degradation of tissue to which we now refer, and its distribution maps out with tolerable accuracy the areas chiefly implicated by these agencies ; and, moreover, the localisation of such wasted areas has an important bearing upon the history of the acute attack preceding the consecutive atrophic change. It is also a very noteworthy feature with respect to the degradations of tissue so resulting, that the peripheral zone of the cerebral cortex is far more prone to implication than deeper regions, at an early stage of the process ; eventually these elements attack the deeper layers and the medullary strands, but their destructive agency is chiefly exhausted upon the **nerve-fibre plexus** formed by the naked, unprotected processes of the cells. The importance of this fact is at once evident when taken in connection with the prevailing view as to the autonomy of the cell and the significance of its ramifying processes ; the cell represents the **sensory unit** of mind, and the processes whereby its connection with similar units is effected represent the **relational element** of mind, the means whereby a change from one state of feeling to another is rendered possible. With the breaking-down of these nerve-fibre plexuses the relational element of mind progressively suffers, the **intellectual vigour wanes;** whilst the **purely sensuous element** may long hold its own.

The nerve-cell itself eventually succumbs to a disintegrating process, possibly secondary to the destruction of its processes ; and (as we have already indicated, p. 537) relics of such cells, in all stages of dissolution, are to be seen scattered throughout the cortex in advanced cases of consecutive dementia. The tissue-destruction is of such a nature as not to lend itself to the processes of repair ; and, in the appearances so presented, we must learn to recognise a *permanent mental enfeeblement.*

From the clinical and pathological aspects of acute and chronic insanity we feel justified in making the following assumptions :—

(1) Acute insanity may be regarded as a very general implication of the sphere of mind, and hence of a wide-spread disturbance of the cerebral cortex.

(2) Its operation is decidedly concentred upon the **motor** or **fronto-parietal** section of the hemispheres, judging from the disposition of morbid appearances and the **resultant atrophy** of the **secondary dementia.**

(3) That in certain forms of mental derangement (the **fulminating psychoses** as they may be termed) approximating to the epileptic and convulsive neuroses in their character, a more **local origin** is often presumable; and in such instances we often find sensory areas peculiarly prone to implication.

(4) That even in the more universal implication of acute insanity, the full force of the nerve-storm falls with unequal strength upon special areas ; as indicated in the uncured wrecks of our asylum

chronics, whose brains exhibit a very variable and unequal distribution of the wasted areas.

(5) In its destructive implication of the cortex, it is the **intercellular nerve-fibre plexus** (the relational element) which primarily and principally suffers.

(6) That the atrophic state induced in the consecutive forms of dementia results in a greatly-diminished brain-weight, as long since indicated by Sir J. Crichton-Browne.*

Let us consider these more in detail. Our studies of the pathology of insanity would impress us with the important principle, that whenever the nervous elements of the cortex are *primarily the seat of disease* originating mental derangement, then the implication of the sphere of mind tends always to be more generally or universally involved; but that where the nerve-changes are secondarily induced as the result of vascular disease, the greater tendency is shown towards a local or partial implication of that sphere. In the former case, the intimate connection of the nervous system may partly explain this more general implication, as it is scarcely possible for any serious affection of any cortical area to be established without involving neighbouring and distant territories closely associated in their functional activities, and, therefore, in organic connection with each other; but, in the latter case, the territorial independence of the arterial supply and the terminal nutrient twigs of the cortex impose, as is well known, a *localising* character upon most of the nervous affections originating in vascular lesion. Hence we find that, whereas in ordinary uncomplicated acute insanity (acute mania or melancholia) the territorial implication is a **very general one,** although invariably expressed at certain sites more than at others; yet, that in certain insanities (*e.g.*, that accompanying general paralysis), special **sites of election** are taken by the diseased process, one area being affected after the other, until ultimately the localising character of the ailment fades into a widespread, general implication. So, again, in alcoholic insanity, the resulting dementia is peculiarly apt to exhibit this partial and **restricted character** (especially in certain forms of amnesia to which we have previously alluded); upon the other hand, the gradually advancing, yet universal implication of mind in the decadence of senile atrophy would imply not as in the former a vascular, but a primarily **nervous origin.**

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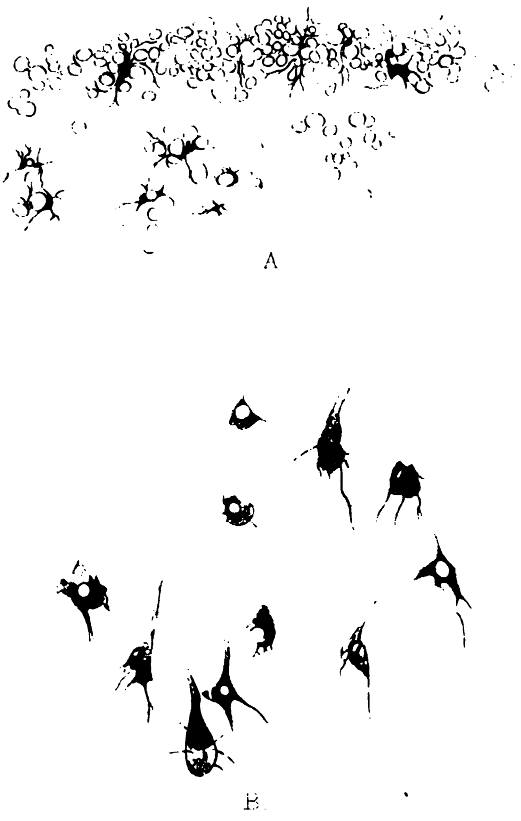
* The statement made by Crichton-Browne is to this effect:—"Consecutive and chronic dementia, a form or forms of mental disease, embracing so many of the inmates of our lunatic hospitals, whose nervous systems have been irreparably damaged by the acute storms of disease, or who have subsided quietly into the depths of fatuous degeneration, is represented in Table vi. by a brain-weight only a shade greater than that of organic dementia; the average for males being 1315·3 grammes, and for females 1159·5 grammes." *Brain*, vol. ii., "On the Weight of the Brain and its Component Parts in the Insane."

Plate XIX



Sclerosis of Cornu Ammonis in Epileptic insanity
A. Peripheral zone in Gyrus hippocampi.
B. Vacuolated cells beneath the above

Bale & Danielsson Ltd, Sc

This tendency of primary nervous implication to share in the universality, which also characterises nervous affections of hæmic origin, should not blind us to the fact already emphasised, that in the widest-spread mental disturbance the morbid implication is always more strongly expressed in certain directions than in others; and that, in a certain sense, we may with propriety speak not of insanity but of insanities, multiform in their nature, and all pointing to certain definite weakened areas in the material substrata of mind. In this sense we have long been accustomed to appreciate Dr. Hughlings-Jackson's dicta when dealing with the reductions of insanities; and in this connection "a plea for the minute study of mania," by Sir J. Crichton-Browne, is well worthy of attention.* It is in this direction that we may yet hope for much enlightenment at the hands of clinical observers upon the question of cerebral localisation, or, at all events, for facts confirmatory of the results of experimental enquiry. Dealing, however, as we do here, with *mental* operations, the alienist has a field before him which extends far beyond the present limits of possible physiological experimentation.

The superficial wasting of the cerebral hemispheres in insanity is far more general and extreme in the fronto-parietal segment of the brain; in fact, as we have before seen, in three-fourths of all cases of cerebral atrophy we find the wasting limited to this division. It is the so-called motor, and intellectual, and inhibitory sections of the hemisphere *par excellence* which suffer most severely as the result of acute insanity and its sequelæ; not the assumed sensory section. The more localised wasting, on the other hand, where limited to individual gyri, exhibits the same tendency to locate itself in motor areas, affecting in order of frequency the centres for the (*a*) lower extremities; (*b*) the upper extremities; (*c*) the face and tongue; whilst the separate frontal gyri come in order of frequency between the two latter. Last of all come the sensory areas of the temporo-sphenoidal, occipital, and angular regions (fig. 24, p. 511).

A very different feature is presented by the localised softenings, due, in far the greater proportion of cases, to thrombosis of the cerebral vessels. Here it is distinctly seen (fig. 22, p. 506) that the sensory areas of the upper temporo-sphenoidal, occipital, and cuneate divisions are most prone to become involved; the motor areas of the ascending frontal and postero-parietal following in their turn. In mental derangements associated with (? determined by) these vascular affections, therefore, we find the most persistent hallucinations of hearing; and this is a suggestive feature taken in connection with the proneness to the implication of the fourth or sphenoidal branch of the middle cerebral, and the occipital division of the posterior

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* *Brain*, vol. iii.

cerebral arteries. Alcoholic insanity, perhaps, affords us the best instances of the kind. What it is that determines the more frequent implication of these arterial channels than the other branches of the same trunk, we cannot at present even surmise.

With respect to the question of localisation as affecting the frontal division of the hemispheres, it will be occasionally found that the atrophic sequelæ of insanity exhibit a very notable **wasting** of the **frontal lobes**; the atrophy to which we allude is so extreme as to give this lobe a peculiar pointed aspect, reminding one strongly of the cerebrum in the rabbit as regards its general outline. We have met with several instances—three especially marked cases—of this extreme atrophy of the frontal lobe. Its importance depends upon the constant association of a definite series of symptoms, which seem to us to have a localising significance and to which we drew attention in a former article on cerebral localisation.* The symptoms to which we refer comprise a peculiar form of dementia, in which **extreme somnolence** prevails, and an utter incapacity for the most **trivial mental effort.** Unlike many dements, their attention can scarcely be even momentarily aroused, and then only to be followed by a lapse into the profound torpor which simulates sleep. This condition of somnolence, lasting day and night, may continue for months, or even years, ere a fatal termination ensues. The subject is a perfect automaton, moves only when pushed along, requires feeding by hand, but swallows the bolus of food when placed in his mouth, and lies in bed torpid and motionless, giving utterance to no articulate sound. In one case—that of an aged demented female, in whom the framework, muscles and integument, testified to extreme atrophy—such a condition was induced during the last two years of her life; but, *at long intervals*, evidence of nerve-instability was forthcoming in the sudden, unexpected outburst of frantic passion, in which she struggled, kicked, screamed, and swore, employing a very free vocabulary of abusive epithets; the outburst would last but for a minute, when the profound torpor again ensued. For months prior to her death she remained bed-ridden and during the whole of the period, except when roused for feeding, in a state of apparent profound sleep. Feeding had to be pursued with care, as she would often neglect swallowing the food placed in her mouth to lapse into her drowsy state, from which she was roused only by shaking and continuous exhortation.

In another typical case the patient was the subject of general paralysis; in this form of disease the symptoms now referred to, and the frontal atrophy associated therewith, are not of very infrequent occurrence; the condition is one of long standing, and must not, of course, be confounded with the temporary stupor of the congestive and apoplectiform seizures incident to this disease.

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* *Brit. Med. Jour.*, vol. ii., 1883.

That extreme atrophy of the frontal lobe may occur in *congenital* cerebral affections without the symptoms here alluded to, is evident from the case of an epileptic lad at the West Riding Asylum, whose frontal gyri presented attenuation to mere riband-like folds; and in whom restlessness predominated. Yet, in his case, intelligence was so far extinct, that he showed no appreciative recognition of any objects around him, and could not be taught to feed or clothe himself, or attend to any of his bodily wants; he mechanically sucked everything placed in his hands; could just utter imperfectly his own christian name. All his senses were intact. Goltz, in his removal of the frontal region in dogs, noted that the senses were intact; there was great irritability and restlessness; they had a stupid, fixed expression of eye; in following a bone thrown before them they apparently forgot their object and passed it by.*

Horsley and Schäfer observed temporary stupidity in monkeys, from whom the prefrontal lobes had been removed; but more to our point at present are the observations of Professor Ferrier, who, upon removal of the prefrontal region in monkeys, noted the following facts—to quote his own words:—

> "Instead of, as before, being actively interested in their surroundings, and curiously prying into all that came within the field of their observation, they remained apathetic or dull, or dozed off to sleep, responding only to the sensations or impressions of the moment, or varying their listlessness with restless and purposeless wanderings to and fro. While not absolutely demented, they had lost, to all appearance, the faculty of attentive and intelligent observation." †

It is impossible, upon reading this description, not to be struck by the remarkable similarity presented, in the mental deterioration of the patients to whom we have alluded, to the animals in whom the prefrontal lobes had been removed.

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* *Pflüger's Archiv.*, Bd. xxxiv., 1884.

† *Functions of the Brain*, 2nd edit., p. 401.

PATHOLOGICAL ANATOMY OF GENERAL PARALYSIS.

Contents.—*The Brain and its Membranes:*—Early Implication of Vascular Tissues—Vital and Mechanical Effects—Effects on Lymph-Connective System—Intra-Cellular Digestion—*Rôle* of Phagocytes, or Scavenger-Cells—Character of Scavenger-Element—Its Vascular Process—Fuscous Degeneration of Nerve-Cells—Three Stages of Morbid Evolution:—Inflammatory Engorgement—Implication of Pia Arachnoid—Nuclear Proliferation on Adventitia—Paralysis of Arterial Tunics—Diapedesis—Exudation—Hæmorrhagic Transudations—Arachnoid Hæmorrhage—Second Stage:—Hypertrophy of Lymph-Connective System—Fuscous Change and Removal of Nerve-Cells—Nature of the Destructive Process—Early Implication of Apex Process—Third Stage:—Fibrillation and Atrophy. *The Spinal Cord:*—Spinal Cases in Four Groups—Evolution of Pseudo-Tabetic and Spastic Paraplegic Forms Pathogenises of Transient Tabetic Forms—Changes in Vascular, Connective, and Nervous Elements—System-Implication of Lateral Columns—Secondary to Cortical Lesions—Respects Systematic Barrier—Chronic Parenchymatous Myelitis—Dependent on Gradual Degeneration of Cortical Cells—Amyotrophic Form—Degeneration of Cornual Elements in Cervical Associated with Descending Lateral Sclerosis in Dorsi-Lumbar Regions—Combined System-Implication of Columns—Pseudo-Tabetic Forms—Ataxic Tabes—Loss of Knee-Jerk—Anorexia—Flashing Pains and Sensory Symptoms—Genuine Tabetic Forms in General Paralysis.

The Brain and its Membranes.—The earliest indication of morbid change is certainly presented by the **vascular tissues**; turgescence of the vessels of the pia, great distension and engorgement of the cortical arterioles, are seen as the apparent result of an irritative process in their tissues. The perivascular lymph-channels are the site of a nuclear proliferation and segmentation of protoplasm, often so enormous as to entirely conceal the enclosed vessel from view. Certain methods of staining and preparing nerve-tissue are peculiarly adapted for exhibiting this change in the early stage of general paralysis; and it may be stated that the usual chrome methods are so prejudicial to the morbid texture, that those who *exclusively* adopt them must have failed, as a natural result, to appreciate the true nature of the morbid change produced, and the very serious implication of the vascular tracts which ensues. This development of nuclei along the lymph-sheath embracing the vessel must be regarded as a genuine inflammatory condition; accompanying it, we observe the usual signs of an inflammatory process, a transudation of the fluid contents of the vessels into the lymph-channel and tissue beyond—a *diapedesis ;* or an escape of amœboid leucocytes from within the vessel through its coats, and collections of hæmatoidine crystals, frequently at the angular bifurcation of the vessels or between it and the perivascular sheath.

The results of this inflammatory process are very damaging to the blood-vessel itself; changes are induced of a **vital** and **mechanical** nature. *Vital*, in so far, that the neighbouring inflammatory state of the sheath appears to paralyse the **tunica muscularis** of the smaller arteries, and the natural elasticity of the vessel becomes also impaired; a relaxation ensuing which favours in a high degree stasis of the blood current, aneurismal distensions, and, on further mechanical obstruction, rupture of the vessel. *Mechanical*, in so far, that the uniform support of the **adventitial sheath** is impaired or lost, or its nuclear accumulations encroach on the lumen of the vessel and compress it; or its transudation-contents in like manner (or otherwise) prejudicially affect the other tunics of the vessel. At a still later period the sheath is yet further damaged by the numerous branching processes of cells extravascular in position, which play so important a *rôle* in the morbid process, and to which we must now direct attention.

In this, the second stage, there proceeds a remarkable development of the **lymph-connective system** of the brain. The cells, which are usually described as "glia cells," or what we have in our anatomical section alluded to as the "flask-shaped elements" of the neuroglia, undergo a wondrous transformation, the real significance of which does not appear to have been hitherto appreciated. We will first describe these elements in their pathological developments; and, subsequently, allude to the important *rôle* they play in the morbid evolution of this disease.* As before stated, these elements are small flask-shaped cells with a comparatively large nucleus at their greater extremity, which latter stains but faintly with aniline-black, whilst the protoplasm of the cell itself remains unstained, and so delicate as to be recognised with difficulty in healthy states. Each has a connection by a delicate process with a neighbouring blood-vessel, and, in frozen sections fresh examined, exhibits several radiate branches so fragile, and excessively delicate, as to be only seen after a keen search, since they remain wholly unstained by reagents (*Pl.* xxvi.). In the morbid change to which we now allude, these flask-shaped cells enlarge very considerably into great amœboid-like masses of protoplasm, often exhibiting subdivision of the nucleus; and, what is of great import, their protoplasm *now stains deeply* with aniline, although not so intensely as do their nuclei. From this extraordinary cell of protean form radiate on all sides numerous branching fibrils, forming an intricate and delicate network around it as a centre, all of which branches, even to their

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* Intra-cellular digestion is now an established pathological fact, and the researches of Metschnikoff have extended largely the *rôle* of certain cellular organisms in the elimination of morbid material. The term *phagocytes*, which he employs for those large cells active in the removal of effete material in the frog and other cold-blooded animals, we have employed when referring to the spider-cell; but we prefer the term *scavenger-cell* for those fixed tissue-organisms which, as we have seen, have an active physiological and pathological *rôle*. See *Metschnikoff's* Original Articles, *Virchow's Archiv.*, vols. xcvi., xcvii.

most delicate subdivisions, are readily stained by the same reagent. These cells have been termed Deiter's cells; they are all characterised by the presence of a vascular process; but well-prepared specimens show us not *one*, but often *several*, such processes distinguished by their greater diameter, their deep staining, and their termination in a nucleated-mass of protoplasm upon the walls of a blood-vessel (*Pl.* xxvi.). The student's attention should be drawn to the fact that in healthy states of the cortex these peculiar neuroglia elements may be readily distinguished from the nerve-cells, apart from their contour, by the fact that their *nucleus alone stains faintly* with aniline, whilst both *protoplasm of cell and nucleus of the nerve-elements stain deeply*; whereas, in the diseased state to which we allude the morbid elements act like nerve-cells, both nucleus and cell-protoplasm, as well as ultimate fibrils, become deeply tinged by the dye, so that, in some cases, they do not look unlike nerve-cells; and a few may even be mistaken for such, until the "vascular process" is detected. This different reaction in the diseased state is doubtless due to the increased and unnatural vitality * of these protoplasmic masses. These lymph-connective elements (normally spread as free cells, except for their vascular branch, throughout the neuroglia-framework of the brain) multiply by nuclear division and segmentation of the cell-mass until their numbers are so prodigious as to rival the densest groupings of nerve-elements in the same region (*Pl.* xxv.; *Pl.* xxiv., *fig.* 2). Their normal off-shoot from the parent-vessel, or its sheath, explains their more dense distribution on either side of the vascular channels; but they may permeate every tract of the cortex, from the peripheral zone to the deepest layer, and are, moreover, often formed deep in the medullated structure of the brain.

Whenever a branch forms a new connection with a blood-vessel, at its junction with the sheath there is invariably found a nucleated mass of protoplasm, often undergoing subdivision, and this process is specialised by its greater size and depth of staining; the other processes are much finer and more delicate, take a more tortuous course, and branch into numerous still finer ramifications (*Pl.* xxvi.). Co-eval with this morbid transformation, we find the nerve-cells present indubitable evidence of a degenerative process (*Pl.* xxiv., *fig.* 1; *Pl.* xxvi.). The morbid condition of the cell has itself been described by some authors as inflammatory in its intrinsic nature (*Mierzejewski*); but, when carefully studied, we wholly fail to recognise an inflammatory condition, we see but the evidence of a true degeneration due to acute nutritional anomalies, and fail to observe any notable difference between the changes through which these cells pass, and those of the cortex in senile atrophy, except in the greater tendency to a **true steatosis** in the latter state; and still less do we perceive the distinction from what is observed in the "**fuscous" change** of the large cortical cells of the **epileptic's** brain (*Pl.* xxii.).

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* Even nerve-cells in certain diseased states stain *more* intensely than in health —*e.g.*, early stage of fuscous degeneration—but subsequently the reaction becomes progressively less intense.

Where the cortex is extensively invaded by the abundance of lymph-connective cells, the nerve-cells will be found to present every stage of degenerative transformation from an incipient change in molecular consistence and coarseness to a broken-down residue recognised only as a faintly pigmented patch, scarcely preserving its outline as a cellular structure (*Pl.* xxiii., *fig.* 3). What is highly important for us to note is the connection betwixt such cells and the neuroglia-elements just described. The processes of these morbid elements apply themselves to the nerve-cells, surround and embrace them closely; whilst the latter are often overlaid by one or more of these spider-like bodies, still maintaining their connection by a *long straight process* with a *distant capillary* (*Pl.* xxv.).

Occasionally, the branches appear directly connected with the nerve-cell, and at their junction a minute nuclear-like mass, more deeply stained, is seen. Wherever we find the nerve-elements much invaded by their strangely transformed congeners, there we observe advanced degeneration of the cell, to describe which in detail would be to repeat the description already given of *pigmentary* or *fuscous decay* (p. 527). It may be stated here, however, that the apical process appears to suffer at an early stage of the disease, and disappears often before the cell itself is very gravely implicated.

As the nerve-cells undergo more and more serious disorganisation, and dwindle away, so these elements of the neuroglia multiply and throw out their protoplasmic extensions in all directions around, tie down blood-vessels, draw the perivascular sheaths by their contraction out of their normal course so that they become (as represented in the figure, *Pl.* xxvi.) pulled in this or that direction into innumerable angular or funnel-like extensions by the attached processes of these cells; the vessels themselves become contorted, and drawn from their normal direction.

Then a further change ensues, the cellular elements appear to reach a limit to their morbid activity, and expend their remaining vitality in a **dense fibrillation.** The protoplasm of the cell dwindles down as these meshworks of fibres form around it, and the nucleus alone remains as a sort of nodal point from which this fibrillated mesh radiates as from a centre, its branches interlacing most intimately.*

* Such isolated nodules of dense fibrillated tissue of almost microscopic dimensions occasionally occur in the cerebellum. Their import has not to our knowledge been previously recognised.

This stage of dense fibrillation and disappearance of the cell-protoplasm is the **third stage** *in the morbid evolution of general paralysis.*

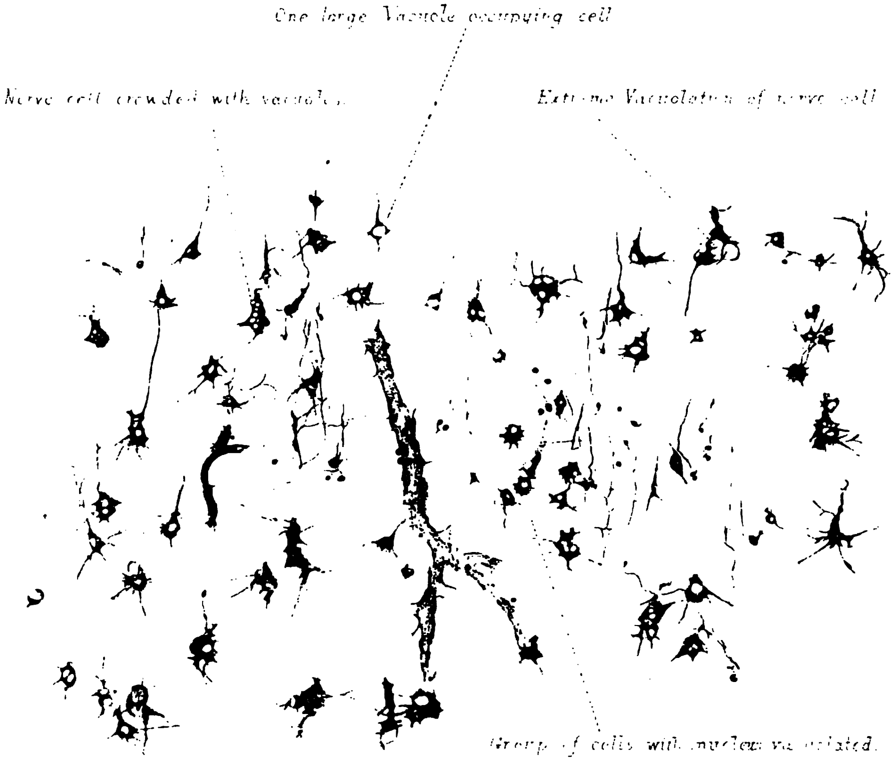
To recapitulate, we have three well-marked steps thus defined whereby we may trace the morbid implication of the cortex.

1. A stage of inflammatory change in the *tunica adventitia* with excessive nuclear proliferation, profound changes in the vascular channels, and trophic changes induced in the tissues around.
2. A stage of extraordinary development of the lymph-connective system of the brain, with a parallel degeneration and disappearance of nerve elements, the axis-cylinders of which are denuded.
3. A stage of general fibrillation with shrinking, and extreme atrophy of the parts involved.

We may now proceed more fully to enquire into the indications afforded us by the morbid changes characterising the above stadia.

Stage of Inflammatory Engorgement.—It is in the vessels of the *pia* that lesions are earliest witnessed, and the lymphatic sheath is that in which the inflammatory change originates. Here it is that, in the earliest stages of the disease observed in the brain of a general paralytic, the initial vascular derangements are first to be noted; and cases proving fatal at an early stage, through the agency of any intercurrent affection, may exhibit (beyond a slight general cloudiness of the arachnoid along the course of the vessels in the fronto-parietal regions, and a very slight increase in the toughness of the membrane) no other naked-eye evidence of disease. The membranes may be slightly more difficult of removal than in health, but show no genuine adhesion to the subjacent cortex. Yet sections of cortex examined microscopically all show a notable increase in the nucleated protoplasmic cells of the *adventitia* of the vessels of the pia, which vessels are also large, distended and often tortuous; together with a general, though slight, proliferation of the most superficial flask-shaped cells of the peripheral zone of the cortex, and the vessels of the intima pia resting upon it. From these cells of the pia long delicate processes are sent out extending deeply down into this layer; and, in fact, simulating in this early stage an appearance often found normally in the cortex of certain domesticated animals (the sheep, pig, rabbit, &c.). That these changes commence in the vascular supply of the pia-arachnoid, gradually extend into the cortex, and eventually penetrate its deepest layers, numerous examinations have established beyond doubt.

In a further-advanced stage of this disease the soft membranes become far more gravely implicated. The nuclear proliferation around the vessels of the pia, their distension and engorgement (from paralysis of the vital contractility of the muscular coat) lead to a very free exudation into the meshes of the pia. The connective trabeculæ lying between the intima pia and arachnoid (which are so especially loose


One large Vacuole occupying cell
Nerve cell crowded with vacuoles.
Extreme Vacuolation of nerve cell
Group of cells with nucleus vacuolated.

Extreme vacuolation of Nuclei of Nerve cells
in second & third layers of Cortex.
[illegible]

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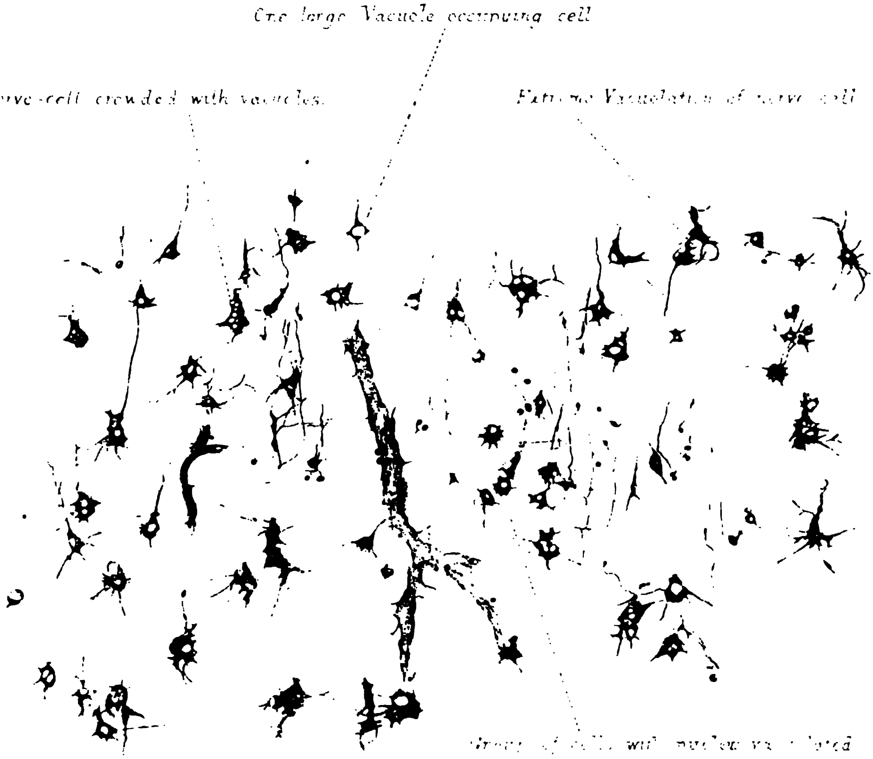
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Group of cells with nucleus vacuolated

[illegible] Nerve cells
[illegible]
[illegible]

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and plentiful within the sulci separating the convolutions) become saturated with a fluid exudate, present a swollen and gelatiniform aspect to the naked eye, streaked with opaque lines, or assume a patchy, or a general and uniformly-diffused opalescence; whilst to histological examination of sections they reveal beautifully-disposed meshworks of connective fibrils, rich in cells, and permeating in every direction the subarachnoid space. Into this space exude the cellular and fluid products of the inflammatory sheath. This tendency to the accumulation of exudate in the subarachnoid lymph-tissue receives a marked increment at a later stage of the disease; for, when atrophic changes occur in the cortex as the result of impaired nutrition and degeneration of nerve-elements, a great compensatory serosity of this region is established, and the membranes become fairly water-logged. The atrophy, which is the result of a genuine sclerous change in the cortex, is necessarily more marked in the sulci than over the summits of the gyri, the area of cortical surface involved in the one case being far greater than in the other, and, in consequence thereof, the gyri become *narrowed* and *attenuated*—the thinning of the cortical layers being the most marked feature.

The vessels in the pia lose the normal support received from the opposed gyri, and, as more compensatory effusion occurs to fill up the space left by the receding brain structure, so the natural support received by their walls becomes lessened, and in the diseased state of their parietes there becomes established a strong tendency to hæmorrhagic transudation, or to actual rupture and hæmorrhage. Such hæmorrhages may be slight and merely punctiform, and are frequently observed; or blood may be transfused into the subarachnoid space and a fibrinous coagulum form upon its meshwork; or, as we often see, a film of blood may be exuded upon the epicerebral surface, between the pia and cortex; or, lastly, the delicate and perforated arachnoid may permit an extravasation on to its outer surface, so that the subdural space may thus become the site of a more or less extensive hæmorrhage. The latter, or so-called **meningeal hæmorrhage,** may be a mere delicate film of blood, or a simple rusty staining of the arachnoid surface, or a thick coagulum of blood extending over the greater part of one or both hemispheres; or a coagulum within a firm fibrinous or organising investment completely encysted; or, again, a thin, but tough, glutinous or fibrinous pellicle, slightly rust-stained, may be peeled off the surface of the dura, forming one or other of the varieties of the so-called arachnoid cysts. These encysted hæmorrhages (which are by no means peculiar to general paralysis, although frequently associated with this disease) appear to be due, in these cases at least, to an initial extravasation caused by the rupture of a diseased vessel in the pia-arachnoid, and to the *subsequent rupture of* *newly-formed vessels* within the organising clot. In none of these cases does it appear to us to have a *direct inflammatory* origin in the membranes. Beyond these extravasations and infiltrations of blood, which invariably occur during an advanced atrophic stage of the disease, we find similar conditions established within the cortex itself. Here, also, the blood-vessels lose the normal support given them in health by the approximation of the perivascular walls, which permit of a limited, but a definitely-restricted, expansion. In atrophy of the cortex, however, these channels become enormously enlarged and filled also with exudate from the contained vessels ; this distension of the perivascular channels favours the aneurismal dilatations already alluded to, and the eventual rupture or transudation of the contents of the blood-vessels into the surrounding space and neighbouring tissue. Hence we get, in all such cases, evidence of extravasated blood in the form of hæmatoidine crystals, which often occur in aggregated heaps, especially in the neighbourhood of degenerating nerve-cells. It would appear that the natural subsidence of compensatory fluid into the sulci, and the much greater recession of the atrophic cortex allowed for by the special position of the walls of the gyri, is unfavourable to the formation of adhesions ; for it is a fact that such morbid adhesions in general paralysis are almost strictly limited to the *summits* of the gyri, where the pia-arachnoid is in close contact and does not permit of the accumulation of serosity to nearly the same extent as in the sulci. As the inflammatory state of the lymphatic sheath of the vessels extends to the deeper layers of the cortex, other grave disturbances necessarily ensue ; and this leads us to the second stage of the disease, which is characterised by the extraordinary development of the lymph-connective system of the brain.

Second Stage.—The implication of the perivascular lymph-channels by the enormous production of protoplasmic masses on their walls, and the blocking of their channels and impairment of the vascular tissues, directly affect the nutrition of the nerve-cells. A granular change in their protoplasm ensues, and **fuscous degeneration** of their contents leads to their ultimately breaking-down into a fine molecular mass of débris. This, together with inflammatory exudates from the vessels, must be removed ; but the lymph-channels are not in such a condition as to ensure this removal of effete material. It is at this juncture that the supplementary lymph-connective element comes into play. In the normal state, maintaining its connection with the vascular walls by its Deiter's process, it either, by circulation of protoplasm or by contraction of the latter, removes such effete material from the cortex. Now, however, these organisms rapidly increase in size and numbers, forming large amœboid masses of protoplasm, which apply themselves to all the degenerative elements around, and by a process of intussusception remove such particles into their interior. For long, these spider-like cells, or Deiter's cells as they have been termed, have been recognised by several authorities in the cortex of the general paralytic, and very varying and conflicting statements have been made respecting them; by some, they were regarded as metamorphosed leucocytes wandering from the blood-vessels; by others, as a simple proliferating connective, which by its pressure and strangulation destroyed the neighbouring nerve-tissues; others, again, saw in them no special connection with general paralysis, since they have been recognised in various diseased states of the brain. It is true that these organisms are met with in other affections of the nervous-system, but simply because they play a most important *rôle* in the pathology of nervous diseases, and it is only when their real functional endowments are perceived that we recognise their important significance in the cortical lesions of general paralysis.

The failure of the usual lymphatic tracts to remove effete matter from the brain reacts by calling forth an increased functional activity in these lymph-connective appendices in the neuroglia, a true functional hypertrophy ensues, and these spider-like elements apply themselves to the task. They become the "phagocytes" or *scavengers of the tissue;* live, thrive, and multiply upon the degenerating protoplasmic masses of nerve-cells and their extensions, and all effete material lying in their neighbourhood is ultimately appropriated to their use. These active scavengers are also destructive of the living tissues; they affix their sucker-like processes to any portion of their structure, and at the point of juncture we invariably see a small speck of active protoplasm containing a nucleus, probably in process of subdivision. Occasionally several of these active elements are seen completely covering a large nerve-cell, which is in an advanced stage of decay, or scarcely visible, forming a mere pigmented molecular groundwork. They are usually noted in great abundance in the deeper half of the peripheral or outermost layer of the cortex, and, being unmixed with nerve-cells, are here peculiarly clear and defined. At this site their destructive agency makes itself felt upon the medullated nerve-fibres, which at this depth run parallel to the surface of the cortex. These, therefore, are the first nervous structures to be involved, and the apical processes of the pyramids are also amongst the first to undergo degenerative change.

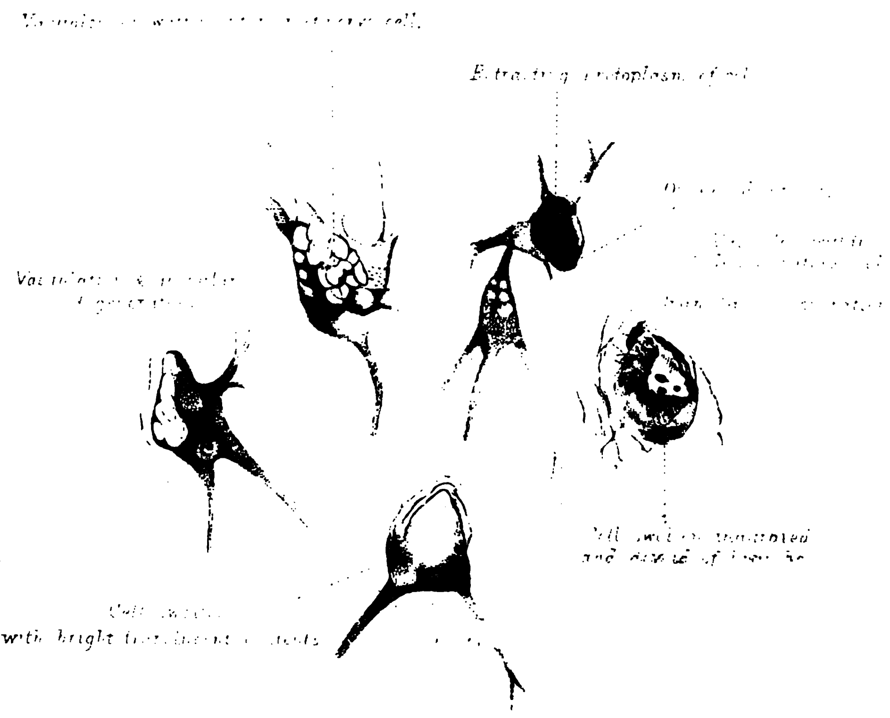
Third Stage.—Like all actively-growing elements, these also have only a limited existence in this condition of morbidly-exaggerated function. The cells throw out innumerable fine processes; and as the fibrillar meshworks increase, so the cell-protoplasm, at whose expense they appear to be formed, dwindles down and eventually disappears.

Hence we have here a veritable substitution of fibrillar connective formed out of the effete material afforded by the atrophic nerve-tissue, a genuine degradation of tissue. The process does not appear to us to be at all akin to the destructive influence of a compression from sclerous invasion; but rather that the presence of the sclerous element is explained by its production out of already-degenerated nerve-elements.

The Spinal Cord.—The spinal symptoms associated with the cerebral disturbances of general paralysis have long been a subject of intense interest to the pathologist, and much diversity of opinion exists relative thereto, less so, perhaps, respecting the *intrinsic nature* of the morbid change as the *mode of implication* of the spinal tissue-elements and the *initiatory* conditions upon which the lesion depends. As is well known, the spinal cord is by no means uniformly implicated in all cases of general paralysis; nor is the selected site of morbid change a constant feature. A large majority of cases of general paralysis pass through the various stages of the disease without any *notable* spinal symptoms, apart from those due to implication of the bulbar nerve-nuclei, until the latest epoch of the affection is reached; whilst in others, from the very outset the spinal symptoms are the most prominent feature of the case. In other cases, again, the spinal symptoms appear to bear a definite relation to the various stages of cerebral disturbance, and vary in their nature *pari passu* with the latter. Thus we may be permitted to group cases usually encountered into four arbitrary divisions.

(1) In the majority of cases we have, as the only evidence of spinal implication, a somewhat general diminution of cutaneous sensibility, associated with a sluggish or greatly-diminished knee-jerk, alternating later on with (or supplanted by) increased knee-jerk, usually as the direct sequel to a convulsive or apoplectiform seizure. Later on, in the disease, *paretic* symptoms may predominate, and contractions be established; but these follow in the wake of pronounced cerebral disturbances (convulsions, &c.), and appear, in fact, to be initiated thereby; whilst the cerebral implication throughout has been all along the more emphasised.

(2) Here there is a second group comprising from the very onset notable *tabetic symptoms*, the cerebral often so greatly in abeyance as to arouse the doubt whether we are not here engaged with a genuine *tabes dorsalis* of local spinal origin. The disturbance of sensation, the abolition of the deep reflexes, the ataxic gait, are all so prominent that we are apt to attribute such symptoms to a primary implication of the cord itself. And yet, in this *tabetic* form of general paralysis, we most usually witness complete subsidence of the special spinal symptoms, the tabetic gait passes off, the knee-jerk returns, and then the full


Vacuolation with ... cell.
Extracting protoplasm of cell
Vacuolation & granular degeneration
Cell swollen vacuolated and devoid of ...
Cell swollen with bright translucent contents

Multipolar ganglionic cells
showing extreme degrees of vacuolation
from anterior Cornu of Spinal Cord
in a case of General Paralysis ×350

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development of the cerebral symptoms is established; or, what is not infrequent, the sensory implication of the cord becomes a motory affection, and **spastic paraplegia** replaces the **anæsthesia and ataxy.**

(3) In yet another series of cases the *motor spinal anomalies* are from the first a most notable feature; and symptoms indicating a symmetrical descending sclerosis of the lateral columns are early apparent, usually as the sequel of convulsive seizures, a mode of implication which appears to us of special frequency in general paralysis affecting those subjects who are addicted to alcoholic indulgence.

(4) Lastly, there are those cases where no spinal symptoms whatever are noticed, the derangements being cerebral throughout (13·6 per cent.).

In explaining the features comprised under these arbitrarily-constituted groups there has been a tendency to regard the later-evolved cerebral derangements of typical general paralysis established in a well-marked *tabetic case* as due to an *ascending change*—*i.e.*, to propagation by direct continuity of diseased tissue; thus making a system-disease of the spinal cord the originating factor of the subsequent cortical lesions of general paralysis.

And, in like manner, the subsequent establishment of motor spinal symptoms (*spastic paraplegia*) has been regarded as a direct transference of morbid implication across from sensory to motor columns of the cord, or to a descending lateral sclerosis having direct continuity with cortical lesions. It appears to us that there is little evidence in favour of such views, which would seem to originate in too servile an attention to the great law enunciated by Waller. The Wallerian degenerations do account for much in the pathological reductions of general paralysis; yet it appears to us to be much strained by efforts to establish its *rôle* when, after repeated attempts made to trace such degenerative continuity of tissue, the best observers have invariably been foiled. It is a notable fact that, despite frequent and most careful examinations of the spinal lesions of general paralysis, we yet fail to trace the continuity of descending changes of the lateral columns of the cord with the tegmental structures of the pons. We are apt in paying too strict attention to the operation of this important law to overlook the transfer of disease to distant parts of the nervous system through implication of higher realms, *not by direct continuity of diseased tissue*, but through the vasomotor agency operative upon nervous tracts in physiological sympathy with their higher centres.

Vascular System.—Nearly all cases present an apparent increase in the number of the vessels of the posterior columns. The appearance

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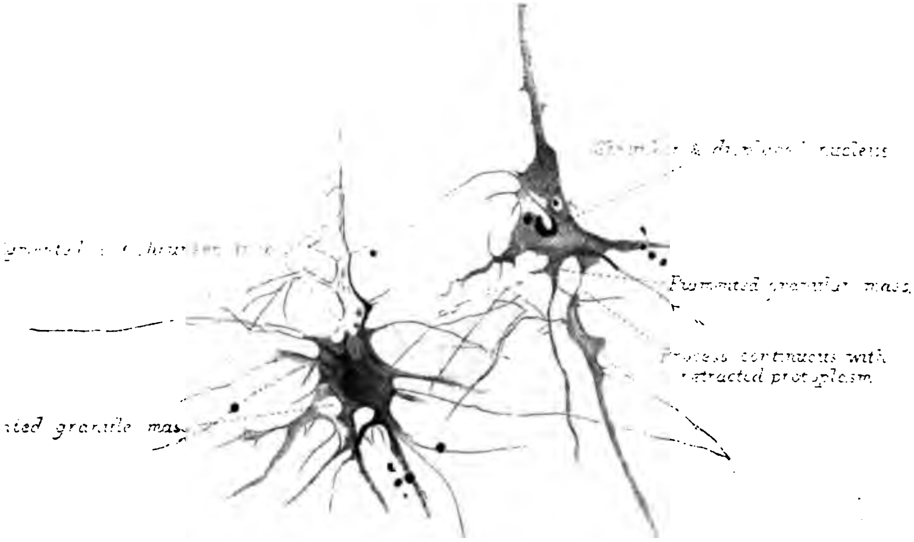
The Spinal Cord.—The spinal symptoms associated with the cerebral disturbances of general paralysis have long been a subject of intense interest to the pathologist, and much diversity of opinion exists relative thereto, less so, perhaps, respecting the *intrinsic nature* of the morbid change as the *mode of implication* of the spinal tissue-elements and the *initiatory* conditions upon which the lesion depends. As is well known, the spinal cord is by no means uniformly implicated in all cases of general paralysis; nor is the selected site of morbid change a constant feature. A large majority of cases of general paralysis pass through the various stages of the disease without any *notable* spinal symptoms, apart from those due to implication of the bulbar nerve-nuclei, until the latest epoch of the affection is reached; whilst in others, from the very outset the spinal symptoms are the most prominent feature of the case. In other cases, again, the spinal symptoms appear to bear a definite relation to the various stages of cerebral disturbance, and vary in their nature *pari passu* with the latter. Thus we may be permitted to group cases usually encountered into four arbitrary divisions.

(1) In the majority of cases we have, as the only evidence of spinal implication, a somewhat general diminution of cutaneous sensibility, associated with a sluggish or greatly-diminished knee-jerk, alternating later on with (or supplanted by) increased knee-jerk, usually as the direct sequel to a convulsive or apoplectiform seizure. Later on, in the disease, *paretic* symptoms may predominate, and contractions be established; but these follow in the wake of pronounced cerebral disturbances (convulsions, &c.), and appear, in fact, to be initiated thereby; whilst the cerebral implication throughout has been all along the more emphasised.

(2) Here there is a second group comprising from the very onset notable *tabetic symptoms*, the cerebral often so greatly in abeyance as to arouse the doubt whether we are not here engaged with a genuine *tabes dorsalis* of local spinal origin. The disturbance of sensation, the abolition of the deep reflexes, the ataxic gait, are all so prominent that we are apt to attribute such symptoms to a primary implication of the cord itself. And yet, in this *tabetic* form of general paralysis, we most usually witness complete subsidence of the special spinal symptoms, the tabetic gait passes off, the knee-jerk returns, and then the full

Plate XXII


Shrunken & displaced nucleus
Pigmented granular mass.
Process continuous with retracted protoplasm.
ated granule mass

Fuscous degeneration of large Ganglionic cells of Motor Cortex. Human ×300.

Bale & Danielsson Ltd. Sculp

each segmented node of the nerve-fibre take the chief part, a process clearly enunciated by Ranvier. If these longitudinal sections are examined in the unmounted state prior to the clearing up with oil of cloves, the fibres are also seen to have freely scattered over them a large quantity of compound granule-cells—another indication of the inflammatory change. These granule-masses are immediately lost upon the use of this clearing reagent, but may be temporarily preserved by mounting in glycerine.

Returning to our transverse sections of these columns, we find the trabecular tissue largely increased, its radiating cells enlarged, and much fine punctated fibrillar tissue (deeply-stained) intervening betwixt the degenerate nerve-fibres, and following out especially the direction of the vascular tracts. The vessels themselves are unduly large, and very prominent in the diseased part; their walls are invariably thickened, the muscular tissue, more especially, being thus increased; whilst the smaller vessels exhibit the change more notably than the larger; in many cases the lumen may be almost obliterated. The lymphatic sheath may be distended, but this change is not so prominent a feature as in the common form of lateral sclerosis from cerebral focal disease; nor, upon the other hand, does it approach to the remarkable change seen in corresponding tissues in the cortex of general paralysis. The vessels themselves usually form centres from which connective tissue radiates into the surrounding nervous structures placed in the axil of the trabecula; the open lumen, the thick wall of the vessel, and its occasionally distended sheath are prominent objects, and the radiate cells around thrust out their processes into the finely-punctated connective in which the nerve-fibrils are embedded. The appearance is almost suggestive, at first sight, of primary interstitial change; but this can scarcely be maintained in view of the fact that the vessels may be traced through healthy tissue (such as pass through the direct cerebellar columns) into the diseased focus, and that only on their arrival in the inflamed zone do they present the morbid appearances described. The same statement holds good for ordinary descending sclerosis, secondary to cerebral lesions; here, also, we witness the implication of the vessel only upon its arrival at the site of morbid activity. Again, we do not meet with the enormous nuclear proliferation upon the walls of these arterioles, such as we found in the cortex; the adventitia is, as a rule, devoid of any undue proliferation. It is not, however, intimated by this, that a true parenchymatous neuritis may not induce such nuclear proliferation by extension of the inflammation to the vascular tracts and interstitial tissue; but, that, in the *absence* of this change, we probably have positive evidence of an inflammatory extension to the blood-vessel not having occurred. A still more important indication of the change being primarily a *parenchymatous neuritis* is found in the tendency of the lesion to assume a genuine system-distribution; and the argument holds good for these changes in the lateral columns of the cord in general paralysis, just as Gowers indicates that it does for the system-disease of *tabes dorsalis*.

Assuming, then, that the changes met with in the lateral columns of the cord in general paralysis are of the nature of a **parenchymatous** rather than an **interstitial myelitis,** and that this change tends to establish a system-disease of the cord, we naturally ask how the change is primarily induced. Why do the nerve-fibres take on the inflammatory condition described? There can be little doubt that the true explanation lies in the destructive and irritating lesions proceeding in their trophic centres in the cerebrum; for we may safely assume that the cortical cells in communication with such motor fibres also exert a trophic influence over them.* The initiatory change—viz., the increase of the nucleated protoplasts of the medullated nerve-tubuli, we do know occurs as the result of its separation from its trophic centre, as by section or other lesion; and we trace in the cortex lesions of motor cells which indubitably should lead to the changes described. It is a significant fact, also, that one of the earliest indications of the change is the extreme vascularity of the tract affected, in itself, possibly, the expression of the trophic disturbance. To summarise these views:—

1. The change is induced **secondarily** to the cortical lesions.

2. It establishes itself after the **Wallerian principle;** does not overstep its systematic barrier, although it may originate simultaneously at several distinct and distant parts of this tract.

3. It reproduces, in varying degrees of intensity, the character of a **chronic parenchymatous myelitis** with notable vascular change.

4. Its intensity never approaches that of the descending myelitis due to large focal lesions of the cortex, and being in its essential nature dependent upon a **gradually-advancing degeneration** of cortical nerve-cells, and not a sudden or gross lesion such as the former, the irritative influence on the cord is greatly mitigated. The changes found in the peripheral nervous system, their vascular tracts, and the muscular system have been well described by Dr. Alfred Campbell.† In his article will be found an exposition of the view of a *primary toxic* origin for these peripheral changes (as opposed to the view now propounded) given in a masterly manner and with good illustrations. Dr. Campbell found extensive changes in the vagi (as did Colella),‡ in

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* See in this connection "Anæmia as a Cause of Degenerative Changes in the Columns of the Spinal Cord," Report in *Lancet*, March 30, 1895. Also "Destruction of Central Grey Matter in Rabbits on Compression of Aorta" (*Ehrlich* and *Brieger*)

† *Op. cit.*

‡ *Annali di Neurologia*, 1891, pp. 115 to 200.

the phrenic, the mixed spinal nerves and their peripheral terminations, the small blood-vessels of the nerve sheath (as did Goodall and Ruxton),* and, to a less degree, the spinal nerve roots, the cranial nerves and their nuclei of origin (*Wigglesworth*, *Mickle*, and others).

Although, in the greater number of cases, the change found in the posterior columns of the cord is limited to the vascular distension above alluded to, and the abundant production of scavenger-cells; yet, in certain instances, we meet with a genuine *myelitis*, the site of which is usually the posterior radicular zone, often extending across towards the columns of Goll. Here, the nerve-tubuli have veritably undergone inflammatory change; and, as will be described more fully in the lateral columns, the medullated sheath is found swollen, faintly stained, the site of nuclear proliferation and disintegration of myeline. Many of these enlarged tubuli show no axis-cylinder or one which is displaced laterally, and the increase of connective along the vascular tracts often leads to a notable diminution and distortion of these columns of the cord. The ordinary grey degeneration of these columns seen in *tabes dorsalis* is not in these cases reproduced, but a much more irritative process, highly inflammatory in character, and closely resembling the sclerosic conditions of the lateral columns with which it is often associated. (*Pl.* xiv., *figs.* 1–3; *Pl.* xv.).

Combined System-Implication of Columns.—Do the changes found ever resemble those of amyotrophic lateral sclerosis? Such cases present a very rapid downward career, which is mapped-out in the earlier stage by successive apoplectiform and convulsive seizures; the latter are usually unilateral, often limited to the facial muscles, and unattended by loss of consciousness. As a sequel to this seizure or "fit," as the friends term it, a loss of power in one or other limb is almost universally found to exist; usually it is the arm that suffers most after these attacks, the grasping power being greatly diminished, and the subsequent changes in the muscular power and nutrition of this member may be disturbed in advance of the lower extremities. These paretic states at first may be very transient, or last a day or more; the locomotion continues unimpaired, the general nutrition of the body may be unaffected, and exercise be taken without inducing fatigue for a period of one or two years subsequent to the onset of the cerebral disturbance. Then, there appear symptoms which inaugurate the advent of organic changes in the cord; the locomotor powers may still be good, and considerable muscular force may be exhibited, but equilibration is distinctly disturbed, and although the patients may be able to approximate their feet in the erect position, and close their eyes with but slight swaying, yet they stagger considerably in attempting to walk in a straight line (heel and toe). The gait gradually indicates advancing ataxy, the legs are thrown out in disorderly fashion, and the tendency to come down on the heel is also recognised. Yet, in lieu of decreased or abolished knee-jerk, we now find either that it is normal in force and range, or that it is greatly increased. At this stage also, we get ankle-clonos in one or other limb as a frequent accompaniment. The tongue now is protruded jerkily, and all its movements are ataxic, the lips may be exceedingly tremulous, but deglutition is unimpaired.

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* *Brain*, 1892, p. 241.

Attacks of maniacal excitement may now precede sudden failures of power in the lower extremities, and we find ankle-clonos and the knee-tap reaction in excess. The arms now rapidly emaciate, and become correspondingly defective in muscular power; in fact, the most marked feature of the case at this period will be this extreme atrophy of the upper extremities, in which the more specialised muscles are not picked-out in the manner of the ordinary *progressive muscular atrophy*, but the large muscles of the shoulder-joint, the musculature of the arm, and the flexors and extensors of the forearm are chiefly involved. No contractures of the arm occur, or myotatic increase, but complete flaccidity, and the legs do not participate in this sub-acute atrophic state. On the other hand, the legs show more marked sensory disturbances, cutaneous sensibility becomes blunted, there is increased swaying in the erect position, the gait may be that of an unsteady jog-trot, or more notably ataxic. Exalted knee-jerk and clonos may still exist; but, muscular enfeeblement now rapidly supervenes; the limbs tend to exhibit spasmodic fixation, but are more frequently kept stiff and rigid by voluntary effort. The patient is now bed-ridden, and at this stage is usually profoundly demented. Implication of the sensory nerve-roots becomes evidenced by almost complete loss of cutaneous sensibility in one or both legs, and is probably, also indicated by a sharp distressing cry often repeated, as if the poor patient were the subject of sudden *lightning-pains*. Ataxy is also now present to a very notable degree, and the knee-jerk (up till this period normal, or unduly exaggerated) is completely abolished; plantar reflex is also absent. By this stage the subject is in a pitiable condition, helpless in limb, utterly incapable of attending to the most trivial wants, exceedingly timid, and the apparent sufferer from fulgurant pains; there is profound implication of the bulbar nerves, deglutition being so far impaired as to make the effort both painful and full of risk; whilst softened food placed in the mouth is apt to be retained as a bolus in the cheek-pouch for hours, unless care be taken. The extreme emaciation of the upper extremities is also attended by rapid atrophy of the facial muscles, loss of all adipose tissues, and a sharpened pinched expression of the features.

Reverting now to the amyotrophic form described, we note first, that the spinal appear consecutively to the cerebral derangements; and, as before stated, are almost invariably ushered-in as the direct results of apoplectiform or convulsive seizures. The resulting paresis is, at first, nothing more than the post-convulsive exhaustion, often seen in epileptics, in whom also the myotatic increase indicated by the knee-jerk and ankle-clonos is often seen; but, eventually, the inco-ordination established, apart from defect of sensation or patellar reaction, indicates a morbid change in some region of the cord, other than that of the posterior sensory roots, and this change is detected *across the columns of Goll, and partly in the post-commissural zone*, the implication of which undoubtedly leads to inco-ordinate action, without further derangement of cutaneous or muscular sensibility. In fact, a morbid basis is established for the muscular excitability indicated by the increased knee-jerk in a finely punctated sclerosis of the lateral columns of the cord, which may be traced from the dorsal cord throughout the lumbar region, but it may not be at all apparent in the cervical region. (*Pl.* xv.) It is to the increase of this sclerosic state of these columns we must attribute the progressive stiffening of the lower limbs, and their exalted muscular irritability. Later on in the history of these cases, the changes noticed in the columns of Goll spread obliquely outwards so as to directly involve the posterior sensory root-fibres, inducing thereby the notable ataxy and anæsthesia of the limbs; but still exhibiting betwixt lower and upper limbs, the contrast of rigidity of the former (associated with no special wasting) and of extreme atrophy, paresis and flaccidity of the latter. As regards the arms, the changes found in the anterior cornua suffice to indicate the cause of extreme emaciation of certain muscular groupings, and their progressive enfeeblement in motor power. (*Pl.* xxi.) The lesions in the multipolar cells of the cornua also, in like-manner, explain the complete flaccidity of this member, for in this region the lateral columns are not diseased. Charcot's view of amyotrophic lateral sclerosis cannot be advanced here; for we plainly see a degenerative atrophy of the cornual elements at a plane considerably higher than any change indicated in the lateral columns of the cord; the latter, in fact, is first seen in the dorsi-lumbar region, *not* in the cervical, whereas the degeneration of the anterior cornua is first seen in the cervical region. That the latter is established by a sort of projection of the disease forwards from the lateral columns is, therefore, here quite untenable, nor, in fact, can any relationship betwixt the two be affirmed; and this accords completely with what we constantly see in ordinary descending lateral sclerosis from focal cerebral lesion, where the lateral columns may remain for eight or ten or more years profoundly implicated, with no obvious change in the cornua. Evidently, then, this disease in the anterior cornua of the cervical and the lateral sclerosis of lower regions of the cord are independent states, mutually related only as regards a community of origin higher up in the cerebral cortex. Why it is that the cornua are affected in the cervical, and the lateral columns in the dorsi-lumbar cord, can probably be explained only by the special localisation and depth of lesion, or degenerative change within the cerebral cortex.

Then, again, as regards the posterior columns of the cord; we find here the frequent vascular change observed in general paralysis, and the affections whereby the cortical lesions tend to project their influence upon subordinate regions of the spinal axis; the vascular turgescence, however, is not so great in these cases as the purely neural change. (*Pl.* xxiv., *fig.* 3.) The change is not one of connective proliferation, of abundant cell-growth of scavenger-elements, or of notably-enlarged vessels presenting changes in their tunics; it is not a vascular nor interstitial connection, but a purely neural change—a genuine myelitis—tending to spread exclusively along the direction of the sensory root-fibres, as indicated in the description above given. No cases, in fact, would better indicate to us the neural origin of ascending changes in general paralysis, and in certain forms of *tabes*.

If, as often happens, the posterior cornua be also implicated by extension of this lesion to the *substantia gelatinosa*, we get anæsthesia of the corresponding limb.

The order of evolution of the morbid changes, appears to be as follows—first, the posterior median and posterior commissural zones are involved, issuing in inco-ordinate gait; next, the lesion tends to spread over the whole of the posterior root-zone, and along the course of its sensory fibres; at the same time progressive degenerative changes occur in the lateral columns in the dorsi-lumbar region. Ere these latter changes are much advanced, amyotrophic change is observed in the upper extremities, revealing the lesion located in the anterior cornua, and, subsequent to this, a rapid ascending change from this site implicates the bulbar nerve-nuclei, and hastens on the fatal termination.

Implication of Posterior Columns (Pseudo-tabetic forms).—We have thus, so far, dealt with a combined system-affection of the cord in general paralysis, where a postero-lateral change predominates in the lower region, and a polar impairment (issuing in progressive general muscular and bulbar atrophy) is emphasised in the cervical regions. Let us now consider, more particularly, the cases where the former exclusively exists. A notable feature in this class of cases is the predominance of sensorial derangements, not as regards spinal symptoms only, but as expressed in cerebral symptoms also. The mental anomalies appear specially to indicate a wide-spread sensorial implication, and the maniacal perversions are characterised by most vivid acute hallucinations, by very painful emotional states, often culminating in attacks of the most acute melancholia. The painful mental states are all associated with well-marked hysteric outbursts so characteristic of this series of cases. It is only in the later stages of the disease, when the dementia is far advanced, that this painful state of mind declines, or rather is replaced by a condition bordering upon idiocy, often with much frenzied excitement.

Another prominent symptom is that of frequent convulsive attacks, which are often peculiarly severe in nature, and leave wide-spread and notable sequelæ, physical and mental. When such a case presents itself, we are struck at the onset by the marked tabetic gait, a feature especially striking if the subject be in a state of excitement. The feet are planted wide apart, the legs thrown out in most disorderly style, and the heel brought down with disproportionate force. The inco-ordination is further increased by closing the eyes, and the patient cannot stand in this position without falling. Yet, muscular power is in no way *necessarily* impaired, and the limbs will resist forcibly efforts to extend them. Since, however, convulsions are very frequent in such cases, we often find a considerable amount of paresis, but this only of a transient nature at first; great fatigue upon slight exertion may be complained of, or the grasping power diminished, as in one of our cases, to 4 kilogrammes. No permanent paralysis is detected in this early stage; but the all-important fact to recognise is the complete absence of muscular atrophy, and the non-implication of the cutaneous and muscular nerves. Yet, simultaneously with this absence of sensory manifestation in lower planes, we may find the sensory tract of the *trigeminus* implicated—*e.g.*, herpetic eruptions and trophic impairment of cornea.

The ataxic gait is, as usual, a more obtrusive symptom than the same impairment in the movements in the hand and arm; yet an attempt to write, to button the coat, to sew, or thread a needle at once makes evident the fact that the inco-ordination of the hands is as gravely impaired as that of the lower extremities. If convulsions occur, they are usually unilateral, or much more marked on one side than the other; they generally leave behind them a hemiplegic state, often with complete hemianæsthesia. The reductions from such convulsive seizures are often most profound and prolonged, the subjects being left for days together in a state of complete stupor; mute, requiring forcible feeding and catheterism, and keeping the mouth full of saliva. Then, as normal sensation is regained and muscular power returns, we may have wild delirious excitement, which may be associated with desperate suicidal impulses.

Repeated attacks of hemiplegia with more or less complete anæs-

thesia of the same side occur, leaving the patient speechless
helpless for days, until eventually the aspect of the case is one of ut
imbecility.. In the intervals, however, between such seizures, he m
still go about exhibiting notably the inco-ordinate gait, but w
normal, or *more often with acutely-exaggerated knee-jerk.* Contracti
of the limbs now ensue, generally limited to the upper extre
ties, and corresponding to the side usually left paralysed af
convulsive seizures; thus, in a case of right hemiplegia with he
anæsthesia following convulsive attacks, the permanent paralysis
contracture is sure to develop on this same side. Ushered-in
slight initial rigidity of the extensors of the forearm and wrist (wh
permit of wrist- and ankle-clonos upon slight flexion), the flexors s
antagonise and contract the arm in the usual semiflexed and prona
position. In this advanced stage there may still be no vasomo
change in the limb, and no indication whatever of trophic disturban
but at a still later stage the skin of the feet may be cold and bluish,
a co-existent *anæsthesia* may be noted in the skin of the calves,
plantar reflexes, however, still remaining brisk. The lower extremi
may show a certain degree of clasp-knife rigidity, or spasmodic fixat
but no permanent contracture; yet, in the latest stage, the repea
convulsive seizures so far exhaust the energy of the motor tract, t
the patient sits squatting in stooping posture, or attempts locomot
on hands and knees. Deglutition may be little impaired except as
immediate result of epileptiform seizures; grinding of the teeth
very frequent accompaniment.

From the first series these cases are, of course, notably distinguis
at the outset, by the far greater obtrusiveness of inco-ordinat
which, at first sight typically tabetic, is subsequently found want
in that implication of the sensory nerve-roots which would rende
a genuine tabetic condition. No disturbance of muscular or cutane
sensibility, however, is discoverable, except as the immediate outc
of a cerebral discharge. Such cases conclusively prove that i
ordination may result from lesions in the regions of the poste
columns other than the posterior root-zone; and that the posterior r
fibres must be implicated to explain any existing sensory anomali
skin and muscles. We find in the cases presented by this series
the posterior root-zone is absolutely free from disease; and that
morbid implication of the posterior columns of the cord is exclusi
limited to the posterior commissural zone and posterior-median colu
(columns of Goll), in cervical, dorsal, and lumbar divisions of the c
this implication of the columns of Goll with a perfectly healthy
of the sensory root-fibres we have repeatedly recognised.* The le

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* See in this connection the microscopic examination of the cord in the ca
H.U., p. 282.

Plate XXIII.

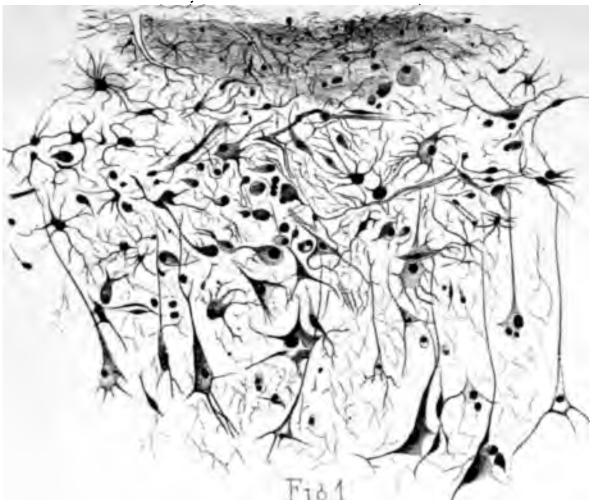

Fig. 1.

Section from the ascending frontal convolution in a case of Senile atrophy. The proliferation of connective cells of the upper cortical layers is seen invading the vascular tracts and nervous elements × 180

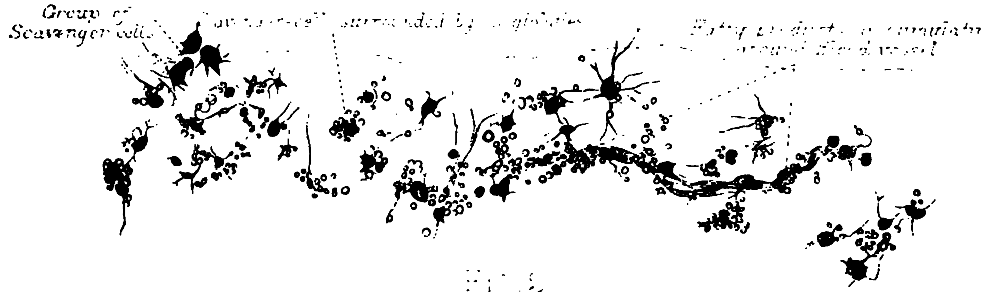
Group of Scavenger cells
Scavenger-cell surrounded by fat globules
Fatty products accumulating around blood-vessel

Fig. 2.

Fatty disintegration of superficial zone (1st layer) of Cortex in a case of advanced senile atrophy of Brain [illegible]

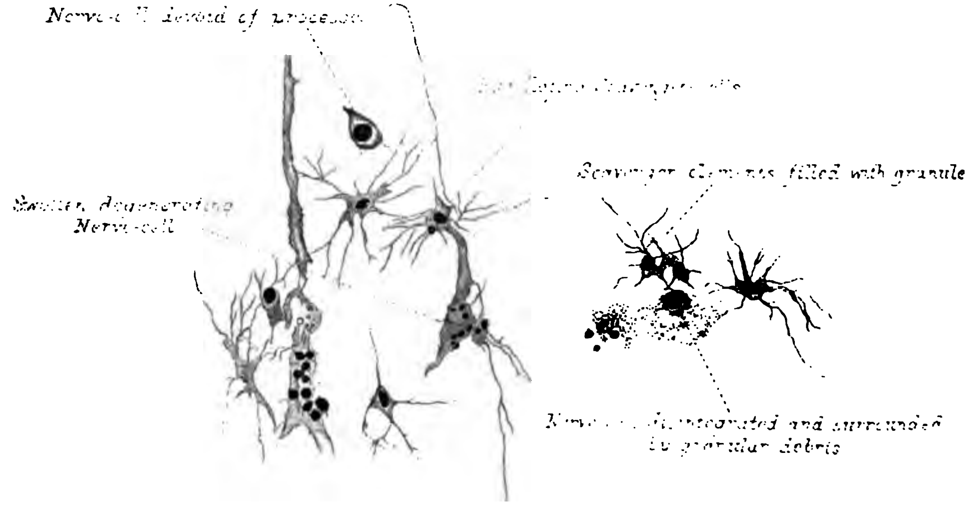
Nerve-cell devoid of processes
[illegible] Scavenger-cells
Swollen degenerating Nerve-cell
Scavenger elements filled with granule
Nerve-cell disintegrated and surrounded by granular debris

Fig. 3

Disintegrating Nerve-cells of Cortex surrounded by Scavenger-cell × 350

observed differs also from that of the former series in being a much more pronounced vascular and cellular change. The vessels of the posterior commissural zone being notably dilated, and extending down the median raphé, are accompanied by a dense crowding of scavenger-cells (the abundant proliferation of which is a striking feature) presenting a coarse trabecular appearance, in which thick-walled vessels with contracted lumen are freely scattered. (*Pl.* xxiv., *fig.* 3; *Pl.* xxvi.) The nerve-fibre does not itself appear implicated as in the former series, and the disturbances in its conductibility are probably the result of the pressure produced by this morbid cellular growth, and the engorged and distended nutrient vessels of this region. The morbid change in tissue follows out very accurately the immediate confines of Goll along the inner half of the wedge-shaped apex, where it lies in contact with the columns of Burdach, respecting rigidly the posterior root-zone, however; but, the columns are throughout their inner half the site of such change, especially along the median raphé. It would appear highly probable that, in those cases where inco-ordination, existing notably *for a time only*, has gradually declined or wholly disappeared, the phenomena may be regarded as *pressure results* which have not proceeded to actual *myelitis*, and in which the scavenger-cells have performed their depurative functions (p. 497) and have been replaced by fibrillated tissue. The muscular tone in all these cases is but slightly, if at all, impaired; and the anterior cornua remain intact, as evidenced by the well-nourished aspect of the muscles late on in the disease; no centric atrophy of the limbs is seen as in the former cases. The general muscular debility and fatigue upon slight exertion, which such cases present, are the outcome of the exhaustive convulsive seizures to which they are so subject, and not of a persistent paralysis or atrophic change in the muscles of the limb. The descending changes in the lateral columns always appear as the sequel of the convulsive seizures above alluded to, and explain the association of exaggerated knee-jerk with the inco-ordinate movements of the limbs; such sclerous change implicates, as before stated, the greater part of the column, respecting, however, the *direct cerebellar* and *intermedio-lateral zone*, it yet creeps forward as far as the postero-external group of cells in the anterior cornu. The change occurring in the posterior median tracts of the cord is earlier in its incidence than this lateral sclerosis, as shown by the much larger development of contractures in the limb after a long persistence of inco-ordination. Then we have to consider the association of the cortical implication with these spinal changes. It is in such cases we get little, if any, indication of adherent membranes, and the atrophic state of cortex will be chiefly located in the upper parietal or postero-parietal lobule. We have elsewhere indicated the association of lesions at this site with tremulous and ataxic movements of the lower limbs,* and it appears to the writer probable, that the implication of the posterior columns of the cord, at the side named, has some direct originating connection with the extreme atrophy undergone in the postero-parietal lobule.

The absence of muscular atrophy, which characterised the former series of cases, is consistent with the immunity of the anterior cornua from morbid change.

Waiving for a time any consideration of a presumed identity in such apparently different neuroses as *tabes* and *general paralysis*, if the question be put as to the frequency of association of the two affections so named, the reply would possibly be in favour of a very infrequent alliance as observed in asylum practice. Nor would this opinion be surprising if we remember that the most obtrusive symptom associated with *tabes* is one by no means essential to the diseased process which is at the root of the ailment. **Inco-ordination**—so prominent a feature in all marked cases of **tabes dorsalis**—need not be present to constitute this disease; and, if **ataxic symptoms** are considered as essential features in *tabes*, then the incidence of such an affection during the evolution of general paralysis would very justly be regarded as most infrequent.

If, however, we adhere strictly to what is accepted as the pathological definition of *tabes*, viz., an affection of the posterior nerve-roots or the peripheral sensory nerves, and accept as its essential clinical feature the abolition of knee-jerk, then we do find evidence in favour of a very frequent association between these affections.

There is substantial evidence (both clinical and pathological) in favour of this severance of ataxic symptoms from the truly tabetic sign—loss of knee-jerk; and the position assumed by Dr. Gowers in favour of such severance appears to us unassailable. There is undoubtedly an ataxic paraplegia *without* the knee-tendon accompaniments of *tabes*; there are, moreover, forms of **indubitable tabes** which exhibit **no inco-ordination.** On the other hand, we have repeatedly verified these data upon pathological grounds, and recognise lesions of a special region of the posterior columns *not implicating* the posterior nerve-roots as the essential condition associated with inco-ordinate movements of the limbs, the knee-jerk being normal or exaggerated; whilst implication of the posterior nerve-roots was invariably associated with the abolition of that reflex phenomenon. Hence, a fallacy is likely to occur in our estimate of the frequency of association of these two cerebro-spinal neuroses; just as in the ordinary form of *tabes* the loss of the knee-jerk is a symptom which may precede the more obtrusive evidence of the disease by many years, being a symptom which is apt to escape detection. It is, indeed, generally revealed at an early date, not from the prominence of any motor inco-ordination, but from the lancinating pains with which it is so frequently associated. Tabes, therefore, in this strict sense of the term, occurs in fully 15·9 per cent. of general paralytics (see Analysis, p. 320), a fairly large proportion; and such cases exhibit certain features which justify us in separately considering the class.*

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* "Localisation in Cerebral Disease," *Brit. Med. Journ.*, vol. ii., 1883.

It is not, however, with these more frequent forms that we are now more immediately concerned, but with the far less frequent association of *ataxic tabes* with general paralysis.

Implication of Posterior Columns (Ataxic Tabes).—This rare form of disease claims our closest attention, not alone from the acuteness of the neuroses, and the wide sweep of the nerve-storm over the most distant regions of the nervous system, but more especially from the emphatic testimony borne by its clinical history to the close alliance (if not identity) of the morbid processes underlying *tabes dorsalis* and general paralysis.

The attack is usually ushered-in by cerebral symptoms which may be purely mental, and of the nature of a maniacal or melancholic outburst; or an apoplectiform or convulsive seizure may occur, such as not infrequently present themselves about the onset of general paralysis. When *mental symptoms* predominate they have usually been of an acute character, tending to melancholic agitation with impulses to suicide or dangerous aggressive violence. We have not observed the wild delirious state seen in the ataxic paraplegia last described. If **congestive** or **convulsive seizures** usher in the affection, there may supervene a transient hemiplegia, more or less complete; and, probably as a sequel to this, we first recognise the inco-ordinate movement of the limbs. We are not in a position to state *when* the knee-jerk declines, from the fact that these subjects come before our notice for their mental infirmity; certain it is, however, that it was lost in all the cases which presented themselves; whether such loss occurred (as is quite possible) before prominent cerebral disturbances existed, or not, is a subject deserving further inquiry. A notable symptom in all these tabetic general paralytics is a severe frontal headache, often complained of to the exclusion of all other symptoms.

The mental state is peculiar in the fact that it is wanting in the *redundant flow of spirits* exhibited by typical paralytics, even where the most grandiose notions prevail. The emotional states associated with ideas of extraordinary wealth, unusual attainments, or wondrous capacities for action, even if they do prevail, are tinctured by considerable discontent, querulousness, and evidence of a general moral decadence. The subject may be distrustful, cunning, treacherous, and exhibit sullen gloom and despondency, or even harbour suicidal tendencies. Dementia may not obtrude itself on our notice until the cerebro-spinal affection is far advanced; and it must be affirmed that cerebral symptoms may entirely fail to manifest themselves, and the patient be sent from under asylum supervision, but suffering from pronounced ataxy of the limbs, and other symptoms of *tabes dorsalis*.

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* The analysis, moreover, indicates, if we include cases where the reflex is not completely abolished, the still higher proportion of 20·4 per cent.

The bulbar symptoms of general paralysis are not necessarily a prominent feature; the pupils may be unequal, and respond sluggishly or not at all to light; the tongue and facial muscles may be somewhat tremulous, but the articulation is often clear and distinct.

On the other hand, the spinal symptoms will be striking features in the case; the patient plants his feet in the manner of the ataxic upon a wide basis of support; when he approximates them, he sways considerably and tends to fall; if he closes his eyes, he must inevitably fall; he fails to walk backwards, and forward progression is accomplished by disorderly thrusts of the leg, first to one side and then to the other, the heels coming down with a forcible stamp. If he be placed upon his back, and be requested to resist extension of the limbs, he exhibits considerable muscular power, and the grasp of the hand may be unimpaired. On percussing the patella-tendons the knee-jerk is found *absent*, whilst the plantar and superficial reflexes may all be present. No anæsthesia or other sensory defect may prevail. As in typically *tabetic* cases, lightning-pains may still further cloud the poor victim's life, and be of so agonising a nature as to render sleep futile, and necessitate frequent recourse to morphia. In all cases it appeared to us that a connection could always be established between the more acute cerebral and spinal exacerbations. The ataxy, in such cases as we describe, is more frequently emphasised in both upper and lower extremities; in uncomplicated *tabes* it is the legs which chiefly suffer, and the arms may, as we know, escape. The ataxy, however, is a symptom which varies in degree from time to time, and is *indubitably worse with coincident mental exacerbations;* the truly *tabetic symptom*—abolished knee-jerk—however, is *persistent, never being regained.* We have known an instance in which both extremities were thus ataxic; notwithstanding the patient was able to write a fairly intelligible letter, although with considerable painful effort and exhaustion. This was the case in the subject detailed by my colleague, Dr. Bullen,* where the patient, tortured by delusions of persecution, spent many hours at the sacrifice of much discomfort in writing down his morbid experiences and recording his accusations against his imaginary enemies. In this case muscular sense was so far defective that he could not touch the tip of his nose with the finger, when the eyes were closed, after repeated trials, nor approximate the tips of the fingers of both hands. It would appear also from the history of this case that the arms were *first* affected (the reverse of what is usual), since slovenly writing first drew attention to the fact of manual inco-ordination.

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* "A case of Locomotor Ataxy followed by General Paralysis of the Insane." *Brain*, April, 1888.

The pains vary much in character and distribution; they are usually sudden, sharp, and lancinating, described as like *electricity passing through a limb;* they may be described as tearing, agonising pains of momentary duration only, or as "**flashing pains**" as one patient described them; or, again, there may be an intense burning pain over a localised spot, as the knee or foot, and occasional "**girdle pains**" supervene. **Rheumatoid** pains are almost always complained of, and the patient will, at times, speak of a spasmodic jerk of the whole arm, due (as he says) to the pain; or from the same cause the leg may suddenly give way beneath him, and he drops on his knees momentarily powerless. Thus, in Dr. Bullen's case "there was momentary loss of power in right leg, with dimness of vision and confusion occasionally," also "hyperæsthesia over the area of Wrisberg."* Priapism and nocturnal seminal emissions occur at an early stage of the affection of the cord, and sexual proclivities are engendered at this period, often colouring the delusional concepts of the subject, his conversation and bearing being suggestive of satyriasis. Impotency follows, and, as indicated by Dr. Gowers, usually when the *cremasteric* and *abdominal reflexes* fail to respond to normal stimuli. Hysteric symptoms often supplement the mental derangements, and a species of insane or hysteric cunning is a prominent feature. In these hysteric outbursts we have seen one patient assault his wife with the most cowardly and uncalled-for violence; others, who have plotted dexterously and with cunning persistence and mendacity to damage the reputation of the nurse or attendant administering to their wants; and others, who have been most foul and obscene in their language. This association of hysteric states in the *tabetic* general paralytic should be remembered, since they are apt also to simulate symptoms and to deceive grossly, if too much reliance be placed upon subjective indications.

Delayed conduction along the sensory strands is known to be of frequent occurrence in tabetic subjects at a certain stage; this we have witnessed in a *tabetic* general paralytic to the extent of *twelve seconds*, which elapsed betwixt pricking the sole of the foot and the registering of the sensation felt.

Gastric, laryngeal, and rectal crises have all been recognised in this neurotic condition, and are so severe at times as to render the patient desperately and determinedly suicidal. In connection with such crises Campbell's statement is of great interest—"In all my cases the pneumogastric nerves were extensively and strikingly diseased, more so than any of the peripheral nerves, and decidedly more than any cranial nerve. My observations in this connection entirely agree with those recently made by Colella (*loc. cit.*), and, in my opinion, it is impossible to attach too much importance to the remarkable singling out of the vagi for such extreme degenerations in this disease."*

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* *Loc. cit.*

Tabetic General Paralysis.—To proceed to the more frequent class of tabetic cases in general paralysis where abolished knee-jerk has been detected, but where *ataxy is absent* or plays quite a subordinate part in the symptomatic *rôle*, we find that a striking feature in the mental disturbance is the almost universal prevalence of melancholic depression, the dejected, hopeless aspect of the patient notably contrasting with the beaming expression of the typical paralytic. A basis for such depression is usually found in a persistent and tormenting sense-hallucination, to which they are prone, and which is not unusually of a sexual nature. One case long observed by us was subject to the persecution of a woman's voice from the neighbouring town, which haunted him whenever he went out of doors, and which prompted him to marry her; the hallucination co-existed even with intense depression and noisy weeping. It is in these hallucinatory states we find an explanation for another frequent association, that of suicidal feelings which peculiarly characterise this class of cases. Almost all such cases have made determined attempts to take their own life by drowning, hanging, strangling, or like desperate means, prior to their admission to an asylum; and their subsequent history is only too confirmatory of this dangerous impulsive tendency. As dementia, however, advances, the more acute melancholy usually declines and is replaced by sullen gloom varied by fitful periods of cheerfulness, in which we perceive the characteristic features of general paralysis—the egoistic state and optimism; the delirious agitation of the purely ataxic forms we do not observe in such patients. Occasionally, but rarely, optimism may be from the onset a prominent feature; there is in such a case advanced dementia. Thus one subject rambled continuously upon his "thousands of champagne, hundreds of thousands of cigars, and his five hundred sons and daughters." The aspect of the patient corresponds to the mental state; it is usually one of gloomy dejection or querulous discontent, in which the vacuous expression of dementia is apparent; the brow is often corrugated from the **persistent frontal headache** so frequent here, and the hair is often rubbed off the frontal region, or off the whole of one side of the head, by the patient's hands;

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* *Loc. cit.*, p. 188.

the skin is swarthy or earthy in tint; there is always a notable degree of **atonicity** in the facial muscles, and, indeed, throughout the musculature of the limbs. Upon the least excitement tremors of the facial muscles are induced, but no twitching; the lips participate in the same unsteadiness, and the tongue exhibits a fine fibrillar tremor; speech is impaired, articulation being slowed, or blurred and thick, or a little quivering; it is never explosive. The oculomotor symptoms characteristic of general paralysis were present in all the cases observed by us.

PATHOLOGY OF EPILEPSY.

Contents.—Modern View of its Nature—An Impalpable Trophic Change—Objections to Methods of Examination—Change in Elements of the Second Cortical Layer—Fatty Change in Nuclei of Nerve-cell—Common also to Alcoholic Insanity—Vacuolation of Nucleus—Ultimate Break-down of Nerve-cell—Implication of Motor-cells—Absence of Vascular Implication—Functional Endowments of Nucleus—Resistance of Cell to Discharge—Nutritional Rhythm—Significance of Size of Cell and Nucleus—Primitive Type of Nerve-cell—Degraded Type of Nerve-cell—Cell-conformation as indicative of a Convulsive Constitution.

The morbid histology of epilepsy is confessedly an obscure question if we confine our attention to those seizures in which coarse brain-disease and naked-eye changes are not appreciable. Upon this point we have the authority of Dr. Gowers to the effect that there is little likelihood at present of our knowledge of its pathological nature becoming more definite, and that—"The changes in the nerve-centres are probably of that fine kind which is revealed only by altered function, and elude the most minute research." There exists a widespread community of opinion that the pathological anatomy of epilepsy, whatever it be, is the expression of a grave nutritional disturbance of cell-protoplasm, a **nutritive disturbance** which need not express itself in palpable morbid change even to the higher powers of the microscope. From this opinion, however, we must dissent; for it appears to us that a morbid appearance of the cortical cell *does exist* of a highly-characteristic nature, when the cortex is the subject of careful examination by the *fresh methods of research.*

Nor does it appear strange that the morbid change alluded to has been overlooked, since the usual methods of preparation are often the least adapted for revealing it; the chrome salts subjecting the cell to very important alterations which obscure the actual state. In the first place, the nervous elements of the cortex involved are the smallest met with; and, in themselves, are not the most clearly demonstrable in a state of health. Again, attention is likely to be distracted by the less-important changes in cells of greater magnitude, where morbid appearances are more pronounced features. In the next place, the tissue-staining is liable to obscure the early appearance of disease unless cautiously performed, and more especially the employment of osmic acid of too high a percentage, or for too prolonged an action.

The change in the cell alluded to is not peculiar to epilepsy; it occurs in other diseases, and especially alcoholic brain-disease, but never to so marked a degree and limited to such special cortical areas as in epileptic insanity. The nerve-elements are not the only ones to present morbid implication, for the connective-element or neuroglia is, as long known, invariably in excess of the normal. To describe the *nerve* lesion first. The small irregularly-shaped nerve-cells, occupying the position of the second layer of the cortex, exhibit a degenerative change which is so far peculiar that the nucleus of the cell is the earliest portion affected, the cell-protoplasm being apparently secondarily involved. The centre of the nucleus is occupied by an extremely-bright, highly-refractile, spherical body—obviously of a *fatty nature*. If the cell be stained by the aniline blue-black the morbid body appears as an unstained bright, spherical bead in the centre of the deep blue-black nucleus; the cell-protoplasm around being in its place differentiated by its lighter staining. In many of the surrounding cells no further change may be observed; but, closer observation shows that either the refractile body has increased so as to occupy the whole available space in the nucleus, the boundaries of which are still mapped out by a deep-stained circle, or that two or more of such bright refractile bodies present themselves within the nucleus, or that the nucleus itself is no longer apparent within the cell, the highly refractile body (in size and outline like the nucleus) being its presumed representative (*Pl.* xix., *B*).

Although the more usual aspect presented is that of a bright spherical droplet of oil, it is by no means invariably spherical, but may assume a crescentic, oblong, or irregular contour. Minute as these nerve-cells are, the strong contrast established betwixt the bright lustrous centre and the deep blue-black aniline stain of the surrounding nucleus, renders the change so distinct that, when once the attention is directed to it, a 1-inch objective suffices to reveal it readily as a wide-spread change in the series of the second cortical layer of cells. It is not here assumed that cells in other layers wholly escape a similar implication, but that, whilst such a nuclear change may be detected here and there in the small and large pyramidal cells of the succeeding layer, it is not an exceptional, but a most frequent, or universal, change in the second layer of the cortex; often every cell within a large field still retaining its nucleus, is seen flashed within by this bright morbid spectrum (*Pl.* xix., *B*). When the change has progressed so far that one-half of the nucleus is occupied by the morbid substance, the former appears to have lost its selective capacity for the aniline reagent, stains poorly, and is but faintly differentiated from the enclosing cell; and, as the fatty change proceeds, any remaining nuclear mass presents such a delicate stippled shading that it fades off into the cell-protoplasm, and is with difficulty distinguished therefrom, or is wholly lost to view. It is interesting to observe the persistence shown by the nerve-cell despite the degenerative change in its nucleus, and it is only later on in the stage of dissolution that the cell-protoplasm betrays evidence of degeneration. That the cells ultimately break-down is sufficiently evidenced by the paucity of elements in this layer contrasted with what is seen in the healthy brain, and by the abundance of fragmentary residue left by the process of disintegration at this level of the cortex.

The more advanced stage of this fatty nuclear change reveals a **vacuolated** condition of the cell, which becomes even a more striking feature than the simple fatty change. This vacuolation is evidently attributable to the bursting out from the cell of the globular bead of fatty substance, leaving the cavity containing it as a very conspicuous object of sharp-cut marginal contour. Usually the cell maintains its original contour, whilst a large cavity occupies its centre, as large as is consistent with the capacity of the cell, so that a perfectly spherical outline is maintained within an angular or pyramidal boundary, the merest rim of stained protoplasm (thickest where the processes emerge) bounding this cortical vacuole (*Pl.* xx.). At times the escape of these contents involves a large margin of the cell, rupturing and destroying its lateral, or its basal, periphery; still the remaining protoplasm elsewhere maintains a rigid skeleton of the original cell, so that little real distortion of the less-affected portion of the cell exists. The evidence of morbid change in the surrounding protoplasm of the cell exists not only in the rigid retention of the form of the enclosed cavity, but also in the presence of pale spots indicating the degeneration of its mass, which are, however, of far less lustrous aspect than the nuclear contents. Scattered amongst the less-diseased cells of this layer we find angular fragments of destroyed nerve-elements, or sheaves of apical processes completely dissevered from any relict of cellular structure. This extreme degree of change, now described as vacuolation of the cell, may occupy the whole of the second layer of the cortex; but, in certain cases, it has been found to affect every layer down to the *spindle-series of cells inclusive.* When the larger cells are the subject of this change, the cell-protoplasm presents aggregated globules of morbid material, obscured by the deeper staining of healthier protoplasm; yet, pale by contrast, it gives the cell a peculiar rugged mulberry-like aspect.

The large "ganglionic" cells suffer very unequally in different subjects and at different sites. In early stages of implication they appe swollen, and take up an intense staining of their protoplasm, so as obscure their contents in aniline blue-black preparations. Such cel in mounted preparations present an unusual relief, with clear-c contour, very unlike the same cell in a further advanced stage degeneration, and are much more sharply defined in this state than health (*Pl.* xx., *deeper layer*). Pigmentary degeneration of a limit portion of the cell may be seen, whilst in the darkly-stained protoplas three or four paler spots are seen, somewhat refractile and gleami through the superimposed protoplasm. Many of such large cells a swollen and globose, maintain their lateral and basal processes, b have no apical process, or merely a stunted one attached; they a uniformly stained of a pale tint throughout, the nucleus having d appeared. When still further degenerated these cells present a blurr outline, as if from fatty liquefaction of their contents; or an extreme faint ghost-like representative of the cell alone remains.

With this fatty, nuclear change and vacuolation of the cells of t superficial cortical layer, we observe no associated vascular chang the vessels may be somewhat coarse, and distended more than usu but no extreme alteration is observable in the tunics of the vessel, course excepting such as may be attributable to other agencies, such the senile or alcoholic degenerations, or the complication of tubercle of syphilis. Nuclear proliferation along the adventitia is rarely se in epileptic insanity. In like manner, we do not meet with t presence of *spider-cells*, which permeate the cortex and medulla whe vascular lesions affecting the blood and lymph-channels prevail. Thu in the morbid anatomy of epileptic insanity we find a special freedo from nuclear proliferation, from vascular degeneration, and fro hypertrophic states of the lymph-connective system, which obtru themselves in alcoholic cases and in the subjects of general paralysis.

Pathology.—The essential nature of epilepsy is that of an abnorm discharge of nerve-force from the higher cerebral centres in the corte an "occasional, sudden, rapid, and excessive discharge" (*Dr. Hughlin Jackson*). It matters not, for the essential character of this affectio whether the phenomena are sensorial almost exclusively or motori whether the sphere of mind is specially involved, or whether there the fullest development of the epileptic spasm; the essence of t disease consists in this excessive *local* discharge.* A nutritive irrit bility underlies the morbid activity, and invariably expresses itself some one or other morbid change recognisable in the structur elements of the cortex. As we have seen in such cases of epileps where mental disturbance predominates and actual insanity co-exists, we have a notable affection of a special series of cells, not exclusively seen, however, in this disease, for it likewise prevails in other convulsive affections, such as chronic alcoholism wherein spasmodic discharges of nerve-energy are frequent.

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* On the origin, essential nature, and conditioning factors of the nervous d charge see a masterly analysis in Dr. Charles Mercier's work—*The Nerv System and the Mind.*

The extensive nuclear degenerations which we have described must issue in the death of the cell. We know little, for certainty, as to the functional endowments of the nucleus, but we may recognise its presence in all conditions of active growth and functional life in the cell, whether it be a nerve-cell or element of other tissues, including the phenomena of karyokinesis. With its atrophy and disappearance we find associated declining functional activity and ultimate degeneration of the cell itself. We have seen elsewhere that there is much reason for regarding the cells which prevail in this layer of the cortex, as pertaining to the *sensory* type of nerve-element, and that a functional connection subsists betwixt them and the large *motor* elements distributed at a lower level; in fact we may, perhaps, regard these individual layers as constituting a highly-complex sensory-motor arc, of which they are the respective poles. What is the functional relationship existing between these elements? That these presumed sensory units have an inhibitory control over the subjacent elements, and that, lacking such control, their discharge will be subjected to the periodicity of the nutritive rhythm is very probable. The changes presented by the cortical nerve-cells have long led us to regard the nucleus as subserving an important *rôle* in the functional activity of the cell; that its displacements, distortion, degeneration, enfeebled vitality,* and its absence are constant accompaniments of cerebral disturbances characterised by *loss of inhibitory control.*†

From this point of view, we have been accustomed to regard the proportionate size of nucleus to nerve-cell as indicative of the inhibitory controlling capacity of the cell in question—its *own resistance to discharge.* Hence, these minute elements with large nuclei in the second layer would possess a far higher degree of resistance to nervous discharge than those of lower levels, in which the nucleus bears a far smaller ratio to the surrounding cell-mass. Thus in these higher levels nerve-discharge would be impeded, and the *resistance* and *time-element* characteristic of the mental operations would come into play. Certain it is that in such cases where nuclear degeneration has proceeded far in this layer, there is a motor and mental instability characterised especially by periods of nutritional rhythm. In like manner, the cells of the motor area are proportionately *large*, and subserve the function of storage of motor energy; but their nucleus is small in proportion thereto, and their resistance to discharge consequently slight, their functional equilibrium is more readily affected; their greater mass requires augmented nutritive resources to reinstate them subsequent to their discharge (*Ross*).

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* As probably illustrated in its feeble staining to usual reagents.

† Nor is this supposition opposed to the results of Kussmaul and Tenner upon the effects of sudden loss of large quantities of blood. Suddenly-induced anæmia by withdrawing the requisite pabulum would directly affect the nuclear centres of cell-life, which are recognised as actively operative in the nutrition of the cell; the withdrawal of such pabulum would be equivalent to a total arrest of such function, to the virtual paralysis resulting in the discharge of nerve-energy from the cell expressed, on the mental side in loss of consciousness, and on the physical side in general convulsions.

It has already been indicated in discussing the etiological relationship of epilepsy that *heredity* plays a prominent *rôle*, and that epilepsy, direct or collateral, occurs in a large proportion of cases; with these are associated ancestral intemperance, which likewise is an important factor. It becomes, therefore, a question worthy of consideration whether we have here to recognise in the structural modification of the cell the physical basis of such hereditary transmission; is it probable that the nuclear and cellular change bears the imprint of ancestral vice? That the inflated spheroidal cell of epileptic idiots is a distinct reversion (or, at least, an undeveloped stage) is doubtless true; not only does its conformation indicate its lowered type; its degenerated protoplasm a sustained nutritional anomaly; its paucity of branches a restricted relational element of cell-life; and its nuclear change in form and position some vital peculiarity inconsistent with the normal activity of the cell; but we also have evidence of reversion in its case, in the appearance of such cells (*i.e.*, of inflated spheroidal elements with few processes) in some lower forms of life, and we have elsewhere indicated their existence as a normal element in the cortex of the ape.* We see no reason, therefore, for doubting that when such cells occur in the cortex of a class who also bear the history of ancestral vices, such as epilepsy and drink, they are the expression of a reversion to a more primitive type so induced.

Here, however, we must distinguish betwixt idiopathic epilepsy in the adult and those forms which are clearly due to gross central change, or such cases where epilepsy is but the accidental accompaniment of developmental arrest. The onset of epilepsy in early life is recognised as highly ominous to the mental well-being, and it is undoubtedly an established fact that, although in adult life in exceptional cases, epileptic seizures may co-exist with great intellectual vigour, yet its occurrence during periods of active cerebral development in infancy and youth is attended by a profound change in such activities, and usually in their total arrest. This fact is often expressed in such terms as to imply apparently that the "fits"—*i.e.*, the convulsive seizures themselves—are the agencies whereby the cerebral activities underlying mental evolution are injuriously affected. It must, however, be borne in mind that the convulsive discharge in itself is *not* the factor in the arrest, but simply betrays the nutritional impairment (in itself the origin of the convulsive discharge, and, at the same time, of arrested evolution). It is in the structural peculiarity of the cell that we must learn to recognise the origin of the convulsion, and of the stunted mental development which such vicious conformation symbolises.

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* *Trans. Roy. Soc.*, *loc. cit.*

On the other hand, in forms of idiopathic epilepsy arising subsequent to the attainment of adult life, the more striking feature presented to our notice is the *degradation* of mind—its gradual obnubilation by progressive dementia. Are we prepared to recognise such distinction in the histological elements of the cortex? We think there can be but little doubt that in the latter cases (*dementia*) we simply witness a degenerative affection of the nerve-cell, which, *apart from this*, betrays evidence of a full developmental constitution. In the former (epileptic idiocy), however, we find an altered type of cell, a limitation of its connecting meshwork, and a confirmation so decided as to at once indicate the distinction. Yet, underlying both forms, we still recognise that disparity betwixt nucleus and protoplasm, and the displacement or degeneration of the former, which to us appears to bespeak a *convulsive constitution*.

PATHOLOGY OF CHRONIC ALCOHOLISM.

Contents.—Morbid Changes in Cerebral Vessels—Scavenger-Cells in Outer Zone of Cortex—Sclerosis of Outer Zone—Amyloid Bodies beneath Pia—Implication of Motor and Spindle-Cells—Significance of these Changes—Deepest Layers more generally Involved—Early Vascular Implication—Aneurysmal Bulgings—Atheromatous and Fatty Change—Pigmentary Degeneration of Motor Cells—Scavenger-Elements in Spindle-Layer—Degeneration of Medullated Nerve-Fibre—Spinal Lesions—Vascularity—Hypertrophy of Tunica Muscularis an Inconstant Feature—Relationships to Chronic Bright's Disease—Sclerosis of White Columns of Cord—Spinal Degenerations in Typical Case—Implication of Clarke's Column—Immunity from Multiple Neuritis—Neurotic Heritage—Chronic Endarteritis—Fatty and Sclerous Tendency—The Brain of the Criminal Class—Exceptional Resemblance to General Paralysis—Coincidence of Grandiose State and Delusions of Persecution—Inconstant Vertical Implication of Cord—Constitutional State that of Chronic Bright's Disease—Exceptional Transition to General Paralysis—Significance of Arterial Changes—Affection of the Visceral System.

The vessels dipping into the cortex from the pia are of undue size, coarse, frequently tortuous, and their coats are in advanced stages of atheromatous and fatty change. The nuclei of the adventitial sheath are somewhat numerous, are freely proliferating, or their protoplasm is in a state of fatty disintegration (*Pl.* xxiv., *figs.* 1, 2.) Far the more prominent feature, however, is the abundance of *scavenger-cells* which pervade the upper or outermost region of the peripheral zone of the cortex lying immediately beneath the pia; these nucleated protoplasmic bodies are everywhere seen, their branching processes forming a dense matting which converts the outermost fourth of this cortical layer into a closely-felted substance of minute meshes, the aspect of which differs strikingly from that normal to this region (*Pl.* xvii., *fig.* 1). Wherever a blood-vessel passes downwards through the cortical layers, these scavenger-cells are more numerous, following the line of vascular channelling, and so dipping down into the nerve-elements of the second layer. The appearance forcibly reminds one of the increase of connective passing along Glisson's capsule in a sclerosic state of the liver.

This felted structure is always most dense immediately beneath the pia, where it is so far condensed as to take a deeper staining of the reagent quite recognisable to the naked eye. The depth of the whole peripheral zone is also perceptibly diminished, the outer fourth being distinctly mapped-off from the rest by its deeper tinge. We meet with this development in different stages; occasionally the cellular element predominates—young scavenger-cells are numerous, their fine extensions being widely scattered and sparse; in other cases the cells are found of larger size, forming plump, amœboid elements, from which radiate processes pass into a fine meshwork around; still later, the protoplasmic masses have dwindled down or totally disappeared, leaving simply the dense, felted, fibrous structure profusely besprinkled with the still remaining nuclei (*Pl.* xvii., *fig.* 1). Beneath the pia, betwixt it and the surface of the cortex in the so-called *epicerebral space*, we often find a vast quantity of amyloid bodies, and the fact that these are abundantly recognisable in *fresh sections* from *frozen* brain is sufficient refutation of the assumption that such bodies are not of morbid nature, but artificial products of alcoholic reagents used in preparation. Here and there along the walls of a blood-vessel a little heap of proliferating nuclei is seen, from which fibrous extensions pervade the cortex on all sides, giving the vessel a peculiar spinous aspect.

The perivascular space is also seen distended by numerous lymphoid elements, and the nuclei of the sheath are often mapped-out by a linear series of oil globules which alone remain to represent the degenerated element. Critically examining the second and third layers of the cortex, we find no very prominent lesion—a few of the lower pyramidal cells may be degenerate—but, until we reach the fifth layer of *motor cells*, no very obvious change is apparent in most cases (*Pl.* xxiv., *fig.* 1).

These large cells, however, are in an advanced stage of fatty change, and together with the layer of spindle-cells immediately beneath, are undergoing extensive disintegration and absorption (*Pl.* xxiv., *fig.* 2). Can we explain this apparent anomaly of the escape of the superjacent layers of nerve-cells, and the extensive implication of the outermost and deepest layers betwixt which they lie? A special selection of certain layers by the morbid process appears to be evident here; and may be a fact of great significance.

In the first place, we must call to mind the fact, that the outermost cortical layer represents the apical distribution of the large, deep-seated cells which have been presumed to possess motor endowments; and that their poles, therefore, are (in the early stage of general paralysis, as well as in alcoholism) affected by the sclerosic change proceeding in the outer layer of the cortex, and that these cells are, therefore, affected by a degenerative change ere the morbid process extends deeply into the small elements of the second and third layers. But simultaneously with this an invasion of cortical-elements also takes place from below—*i.e.*, from the medulla of the gyri, and this morbid process spreading upwards involves both spindle and motor elements successively.

The cerebral cortex presents, therefore, in such cases very notable morbid change; and one specially characterised by the greater concentration of the lesion in motor realms of the hemisphere, as well as by a somewhat definite restriction to certain layers of the cortex, to the exclusion, more or less, of the other layers. The **deepest cortical layers** are those more especially affected; cases being met with where the uppermost layers show no morbid indications whatsoever.

The vascular, nervous, and connective elements all participate in the change, and it thus becomes of interest to learn which of these tissues is primarily involved and, therefore, plays the more important *rôle* in establishing the pathogenesis of chronic alcoholic insanity. A careful study of a series of such cases would lead one to infer that the **vascular** is the first tissue involved in the morbid evolution. The long, straight vessels of the cortex are peculiarly liable to these changes, and where they dip down deep into the spindle-series of cells, such vessels present gross lesions of their tissues, as also of the immediate neighbourhood around.

The vessels themselves are enormously and unequally distended, showing numerous ampullæ or aneurysmal distensions, usually fusiform in character, their tunics crowded with nuclear proliferation. Carefully-prepared sections of frozen cortex often appear riddled by a large number of circular holes, with sharp-cut edges, as if punched out of the brain-tissue; or by long fusiform channels, the site of diseased vessels which may have dropped out; or still convey distended vessels, the walls of which are mottled by atheromatous change, whilst a peculiar albuminoid material (unstained by aniline) fills their cavity or is effused around their ruptured orifices. The nervous, as well as the connective, elements of the upper three or four layers of the cortex may exhibit no morbid change, but at the site of the large, so-called *motor cells*, constituting the clustered groups of the central gyri, we discover a notable degeneration. These great nerve-elements are much swollen and rounded in contour, and, in lieu of their usual extremely delicate protoplasm, present a rough granular aspect internally, which often takes an intense staining from aniline, leaving a portion, however, quite unaffected by the reagent and of a coarsely granular and often yellowish hue (*Pl.* xxiv., *fig.* 1). Such cells are frequently seen deprived of their apical processes by a veritable degeneration. At its connection with the cell itself this process may be greatly and irregularly swollen and pigmented, beyond which a sudden attenuation occurs, and, after a slightly-contorted course, it disappears entirely (see several instances in *Pl.* xxiv., *fig.* 1). Another appearance universally presented by these degenerate cells is the abnormal, coarsely-defined boundary-wall of the cell, which, as we know, does not exist as a separate constituent in the normal cell of health, or, at all events, cannot be differentiated from the protoplasmic contents in fresh-prepared sections from frozen cortex. The formation of this cell-wall, betwixt which and the enclosed protoplasm a mass of pigment collects, the former shrinking as the latter encroaches upon the cell-contents, is a constant feature in all cases of alcoholic degeneration of the cortex; it brings the cell into a peculiarly notable relief, which is observed in other degenerative affections of the cortex. These large degenerate cells have usually several short, stunted, and swollen processes to which nuclei adhere. Three-fourths of their cavity may be occupied by coarse, granular, golden pigment, and the stained protoplasmic residue exhibit a few glistening refractile oil-globules, or one large circular cavity (vacuole), from which such oil-globules have forced their way out, the protoplasm in such a case not filling the vacuum.

Down in the lowest layer of the series—the spindle-cell formation—we come suddenly upon large developments of scavenger-cells, which above this level were not apparent. Such elements, characterised by their spider-like appearance, are scattered profusely upon the coarse blood-vessels of this region (above referred to), and extend their ramifying processes in all directions around (*Pl.* xxiv., *fig.* 2). The spindle-cells, moreover, are themselves covered by heaps of nuclear proliferations which often entirely conceal them from view, so that their position and course are usually mapped-out and alone indicated by these little

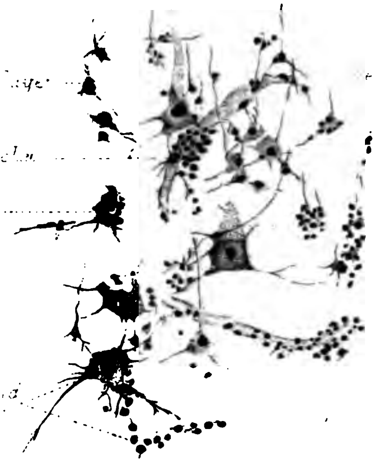

[illegible] granular [illegible] 4th layer
Perivascular nuclei
Perivascular nuclei
[illegible] Nerve cell and [illegible]
[illegible]eneration [illegible] process
Nerve [illegible] disappearing
[illegible] nuclear [illegible]
[illegible] arteriole

Fig. 1.

[illegible] of the [illegible] layer of [illegible] with proliferation [illegible] & [illegible] Insanity ×[illegible]

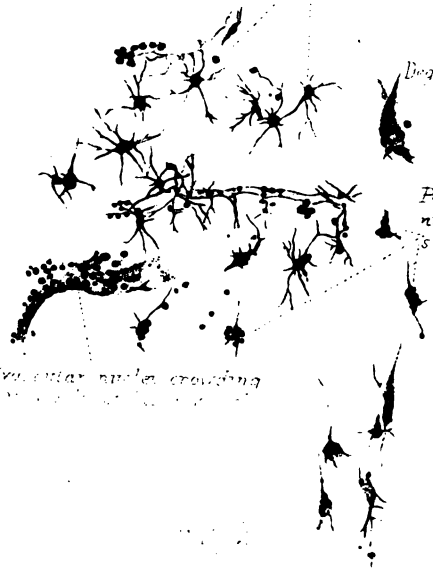

[illegible] proliferating
Degenerating Nerve cell devoid of nucleus
Proliferation of nuclei on fusiform cells
Perivascular nuclei crowding
[illegible]

Fig. 2.

[illegible] spindle cell layer of human Cortex
Chronic Alcoholic Insanity
×420

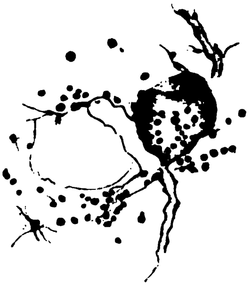


Fig. 3.

Aneurismal dilatation of Perivascular sac
General Paralysis
×210

Bale & Danielsson Ltd Sculp.

nuclear accumulations. One is also struck by their greatly diminished number, and by their frequent pigmentary change where the cell-contents are visible. The conclusion forced upon us by the appearances presented is that they are undergoing rapid degeneration and removal through the agency of the scavenger-corpuscles, which, as previously explained, act in the capacity of "phagocytes," and devour the nerve-elements. In *Pl.* xxiv., *fig.* 1, representing the large motor cells, we observe three large elements with truncated summits undergoing marked degeneration. Above, there is a similar cell, in which the greater part of the apical process is pigmented yellow, whilst at its base a coarse vessel, crowded with a heap of nuclei, is seen. Many small cells are also scattered about, covered with a rich nuclear proliferation. In fig. 2, which represents the same cortex but at a lower level, the spindle-cell formation is seen, sparsely scattered with nuclei, but the site of a rich colony of scavenger-corpuscles. The paucity of the spindle-cells, which, at this site, should be most abundant, is well seen in contrast with a section taken from sensory realms where scavenger-cells are not formed (to the right and below in fig. 2); the cells are not pigmented, but are covered with nuclei. The basal or axis-cylinder process of these large *motor cells* is a very persistent structure in most degenerative affections of the nerve-cell; and, as we have seen, whilst the apical process readily breaks down and degenerates at an early stage, we yet find that this axis-cylinder process persists. If, however, the medullated nerve-fibres passing up from the medullary core of the gyrus into these lower regions of the cortex be examined, a very striking change is apparent. In fresh sections of healthy brain these fibres are not stained by the aniline method; the medullated sheath prevents the reagent gaining access to the axis-cylinder. In certain degenerative conditions, however, a change occurs in the medullary investment, probably of a fatty nature. The medulla is removed or greatly attenuated, so that the axis is exposed and stained readily by this reagent, and then it is apparent that the axis-cylinder is itself greatly swollen and often irregularly fusiform. The identical appearance is also observed in senile decay of the cortex, and here often to a much more striking degree than in alcoholics. Upon the medullated investment, where it appears, spider-cells are often seen abundantly ramifying. The medulla of the convolutions in cases of chronic alcoholism, therefore, presents very notable divergence from the normal appearance, which at once arrests the attention in preparations of fresh brain, stained by the aniline methods, the straight axis-cylinders being prominent objects crowding the field in bundles which can be traced for great distances through the medulla.

On scanning the white matter, we are also struck by the large number of extremely coarse dilated vessels, which afford us evidence also of grave structural change. These nutrient twigs are not only generally dilated, but present along their course frequent fusiform and sacculated aneurismal distensions, often of large size, the coats of which are notably diseased. These aneurismal sacs in many cases will have fallen out of the section, giving rise, as described in the cortex above, to clean-cut circular or fusiform openings, which are often very numerous in such subjects. The sacculated dilatation is often the site of a large accumulation of hæmatoidine granules which crowd its interior, and are scattered profusely over its surface. Occasionally the vessel is seen plugged (possibly by a fatty embolus); the proximal distended part may have ruptured, extravasated blood and hæmatoidine crystals crowding the field around; or a more frequent appearance (seen, in fact, universally over the field) is the distended vessel with the *intima* in a state of atheromatous and fatty change, and the nucleated element of the sheath also undergoing fatty disintegration; the walls covered with young spider-cells, and bristling with their processes on either side (*Pl.* xxiv., *fig.* 2).

Large patches of fatty material containing oil-globules and granules are seen along the coats of the blood-vessels in fresh-stained aniline preparations. As unstained, colourless, and highly refractile spots, contrasting with the healthier and stained tunics around, such patches have a swollen, semi-opaque aspect. All the more degenerate nutrient twigs are the site of a rich colony of scavenger-cells in their various phases of development and retrogression; such elements often look like simple nuclei, until, carefully focussed, their delicate protoplasmic mass and radiating processes are discerned. These scavenger-elements are traced in great abundance throughout the white matter of the convolutions.

Plugged vessels also appear frequently, the tissue on either side being often deep-stained and sclerous in character, and the axis-cylinder in the neighbourhood unduly large and irregularly swollen. The medulla shows a patchy staining of its ground-work to low powers, which on the use of higher objectives is resolved into light, unstained areas having few or no nuclei, and darker stained areas of a fine-punctated aspect (the result of fibrillated spider-cells), amongst which are many nuclei.

Spinal Cord.—Throughout the whole extent of the spinal cord we find increased vascularity, or, at least, a more obtrusive presentation of vessels than is normally observed here. The vessels supplying the posterior columns are those most affected, those of the anterior columns least involved, whilst the lateral tracts suffer almost as frequently as the posterior. These nutrient branches become prominent objects by reason of the great increase in the thickness of their walls—a feature which is exceptionally striking with respect to the smaller vessels between 4 μ and 8 μ in diameter, the open lumen of such divided vessels rarely being over one-third or one-fourth the whole diameter; but vessels measuring, respectively, 18 μ and 36 μ across also have not infrequently a lumen of but 5 μ to 10 μ. This increase in thickness is seen to be due entirely to their muscular coat, which in small vessels of 8 μ diameter will attain the thickness of 2 μ. The increase in the **muscularis** encroaches much upon the cavity of the vessel itself, and the non-elastic **intima** is consequently thrown into a plaited form, or has a condensed deeply-stained appearance mapping it off from the **tunica media**; occasionally the vessel is occluded by this increase in its muscular tunic.

It is not all cases of alcoholism that exhibit this notable thickening of the *muscularis*; for in some we observe far less concentration of the disease upon the vascular supply of the cord than upon the vessels of the cerebral cortex. The following averages represent very conclusively the dimensions of the lumen relatively to those of the arterial tunics in cases where spinal symptoms were a notable feature as contrasted with those in which no special symptoms presented themselves:—

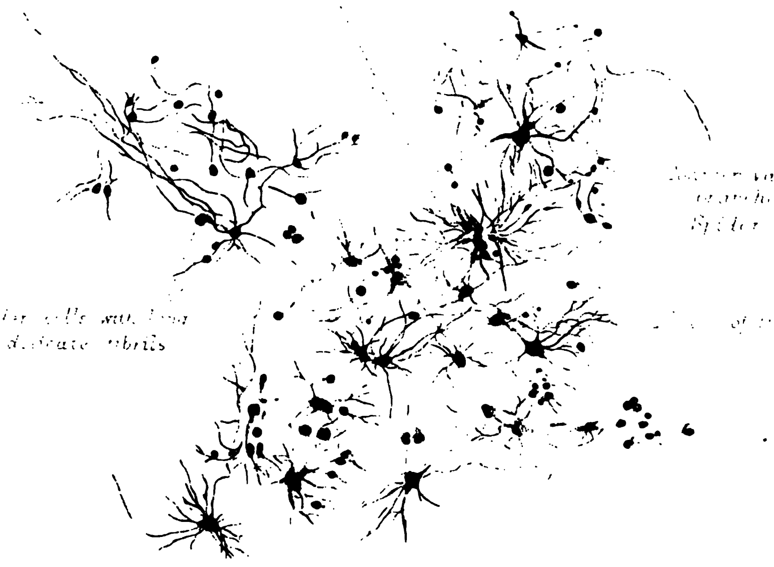
VESSELS IN CHRONIC ALCOHOLIC INSANITY.

Presenting Spinal Symptoms.		Cases Devoid of Spinal Symptoms.	
Whole Diameter of Vessel.	Lumen.	Diameter of Vessel.	Lumen.
18 μ	5 μ	18 μ	13 μ
27 μ	9 μ	27 μ	23 μ
37 μ	10 μ	32 μ	19 μ

The change in these vessels appears to be identical with that increase of the *muscularis* which has now been long recognised in **chronic Bright's disease,** since its discovery by Dr. Geo. Johnston; no notable fatty change implicates the *intima;* the vessels do not here, as elsewhere and in the brain, necessarily show atheromatous degeneration; nor does the adventitial sheath betray evidence of a reactive inflammatory condition. The immediate environment of the vessels shows, in most cases, a normal condition, beyond the prevalence here and there of amyloid bodies in juxtaposition to the vessel. Occasionally these bodies become very profusely scattered throughout the whole extent of the white columns of the cord, more especially around its periphery and following inwards the direction taken by its nutrient branches. In these latter cases we find, however, indications of an inflammatory change—a chronic meningitis; the pia being often greatly thickened, its vessels much distended, and its meshes containing leucocytes and inflammatory products. The connective trabeculæ extending from the pia into the substance of the cord are extremely coarse, and a **diffuse sclerosis** thus originating often affects all the medullated tracts of the spinal cord. Thus, the peripheral zone of the cord is especially implicated; and the sclerosic tissue follows more readily the course of the larger blood-vessels, so that the median raphé of the posterior columns is a favourite site of this sclerous change, which often extends over the whole of the columns of Goll. The coarse deep-channelling by blood-vessels, and the profusion of scavenger-cells, give to the posterior columns a notably morbid aspect. In a typical case examined the antero-lateral columns were extensively implicated; the pia was greatly thickened; and a patchy diffuse sclerosis affected the anterior root-zone, and the lateral columns, together with its *direct cerebellar tract*. The various segments of the cord also showed much irregularity in distribution of the morbid change, and the non-systematic nature of the lesion was clearly demonstrated. The posterior nerve-roots, also, indicate a similar interstitial change; bundles of atrophied nerve-tubuli being seen embedded in much deep-stained connective-tissue. Atrophic changes, also, had involved the cell-groupings of the anterior cornu; and the postero-lateral group in the cervical region on one side was notably affected, few cells remaining, and these degenerated as the result of the invasive sclerosic tissue. The intermedio-lateral group was (in the lower cervical) in a similar state of degeneration on the side corresponding to marked sclerosis of the lateral column.

The **intermedio-lateral group** of cells appears peculiarly prone to degeneration, and other cell-groupings—*e.g.*, the antero-lateral and the internal of the anterior horn on the same side are thus in like manner involved. Clarke's **vesicular column** is likewise liable to implication in these affections. Cornual changes are by no means infrequent, and are of special interest here in relation to the implication of special cell-groupings which are apt to present themselves Thus, in the dorsal region, it is not unusual to find the cells of the intermedio-lateral column of one side plump and healthy; those of the opposite side being utterly degenerated in the midst of a dense sclerous tissue; the same unilateral lesion of Clarke's *vesicular column* may also be observed. In the former class of cases, the naked-eye appearance presents no abnormality in sections across the cord, and it is only in the second series, where wide tracts of connective trabeculæ traverse the columns of medulla, that we appreciate morbid change; which is stil more apparent when the stained section is cleared up and examined That the posterior nerve-roots do occasionally participate in the change has already been stated; but, that the spinal changes originate in the affection of the peripheral nerves is by no means probable; they mus be regarded as coincident affections. Frequent as multiple neuritis i amongst chronic alcoholics of the female sex, we do not recall any cases occurring in alcoholic insanity. That it is occasionally met with

Plate XXV


Spider-cells following vascular tract.
[illegible] vascular branches of Spider cell.
Spider cells with long delicate fibrils
[illegible] of [illegible]

Scavenger elements (Spider cells) in peripheral zone of 1st layer of the Cortex. Human brain
×240

Bale & Danielsson Ltd Sc.

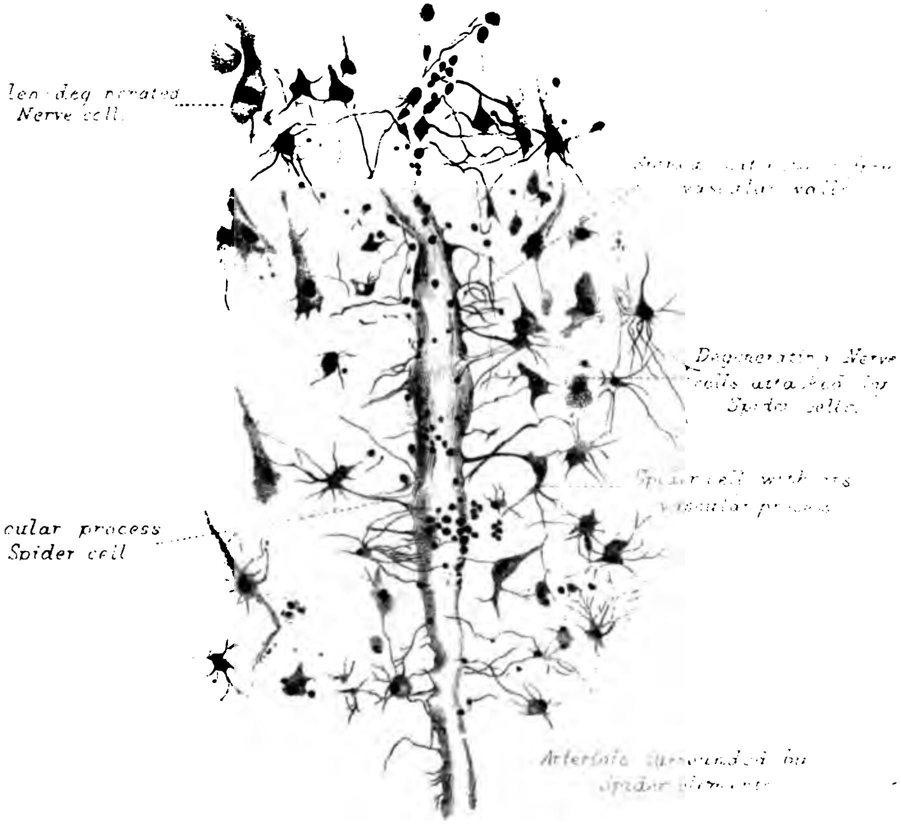
we do not doubt, although the percentage of insane females subject to chronic alcoholism is small; but we must be prepared to regard cases of chronic alcoholic insanity as presenting predispositions which more or less modify the tendency to peripheral implication of the nervous-system. Whatever be the explanation of this paucity of cases of multiple alcoholic neuritis amongst the insane community, certain it is that alcohol in these predisposed subjects *does* tend to concentrate its operations chiefly upon the vascular membranes, first, of the brain and, next, of the spinal cord.

Pathology.—The pathology of alcoholic insanity is but one chapter, though not the least important, in a long history of retrogressive changes to which the whole organism is subjected through the prolonged operation of this agent. Through the medium of the blood-vascular system, alcohol, by its ready absorption and permeability, is rapidly conveyed to the most distant parts of the organism, establishing wide-spread constitutional disturbances; whilst through the peculiar selective capacity of the nervous centres for this poison. it thereupon expends its primary and most potent influence. Although in all cases the nervous centres bear the chief brunt of its attack, it by no means follows that the subjects of chronic alcoholism suffer in the same way. In one, the gastric; in a second, the hepatic; in a third, the renal and cardiac symptoms may come to the front; whilst in others, the nervous centres express the special virulence of the agent in their direction. Undoubtedly a **neurotic heritage** plays a foremost part in thus predisposing to more exclusive determination of the morbid agency upon the higher nervous centres, just as those subjects predisposed to renal degeneration will, on the establishment of alcoholism, display the usual cardio-vascular changes of chronic Bright's disease. Beyond the limits of simple functional hyper-activity of the nervous centres induced by frequent indulgence in alcoholic drinks, its persistent use leads to organic change, first expressed in the vessel's wall by the direct irritating effect of the spirit on its tissue elements. A chronic inflammatory state leading to extensive atheromatous and fatty degeneration of the *intima* is the first apparent effect, associated with which we find parallel changes undergone by the adventitial sheath in the increase and fatty degeneration of its elements. Fat-emboli are frequently established in the smaller cortical vessels during the progress of these changes, and the extensive dilatation and aneurismal states described above are probably direct results of the diminished resistance of the vessel, and paralysis of its muscular coat. An extensive *endarteritis* of a most chronic and insidious character affects the ultimate terminal radicals of the cortex, and, with the pre-existing change in the composition of the blood, leads to the devitalisation of the nervous tissues, undermining the nutritional stability of the nerve-cells. The subsequent change in the interstitial tissue around, and the nervous elements themselves, apparently depends much upon the subject's predisposition, which seems to be the chief determining factor in engendering the fatty or sclerous change which characterises these two classes of alcoholic subjects. In all alike, however, we find the tendency to a degradation of tissue in the replacement of the normal elements by new connective growth; but in some we find a special tendency to extensive fatty change in the nervous centres, so that the parallel degeneration seen elsewhere, as in the fatty or the sclerosed liver, seems to be also reproduced here. It is probable that the fatty change is altogether a *more acute* process, and the sclerosis the result of a *much slower* and more gradual poisoning of the tissues; the fatty change, moreover, is much more liable to be induced in the case of senile alcoholics. We may take it, that the changes observed in the cerebral meninges as well as in the soft investment of the cord, when affected, are undoubtedly indicative of a very chronic inflammatory action proceeding in the vessels of the membranes, and slowly involving the upper cortical strata; for thus only can we explain the frequent association of membranes opaque and thickened, and the permeation of the cortex along the vascular tracts by dense connective networks. Much of the opacity of these delicate membranes is undoubtedly the resulting change of years of excessive indulgence, for it is induced slowly in all cases of long-continued alcoholic indulgence, apart from the establishment of actual insanity; thus, in most criminals, who are notoriously addicted to drinking, we discover such opaque and thickened membranes, and this usually in the postero-parietal regions of the brain (*Henry Clarke*).

Coincident with this implication of the membranes, a similar change is found throughout the nutrient supply of the medullated substance of the convolution, which, as before stated, leads to important changes in the lowermost series of nerve-cells, the spindle-layer and the medullated nerve-fibres themselves at this site. It is obvious, upon examining several cases, that the one site may be chiefly affected to the greater or less exclusion of the other, and that, thus, a sclerous change in the peripheral zone of the cortex may preponderate over any morbid change at a greater depth, or that this deeper implication may be the more expressed feature, the pia-arachnoid being free from notable opacity and thickening. It is more usual, however, to find both areas affected, and this to a profound degree. Certain cases of chronic alcoholism approach, as we have seen, in their clinical features, the history of general paralysis; and when we come to the morbid anatomy, we find the membranes of the brain often presenting similar appearances, both as regards naked-eye aspects and distribution of lesion. The vascular implication, however, is far different, and cannot be readily confused. In the one (alcoholism) the morbid change is centred in the atheromatous state of the inner coat; the numerous bulgings and fusiform dilatation being also highly characteristic of this chronic inflammatory implication. The outer or adventitial investment does not show the *enormous* nuclear proliferation which is so notable a feature in general paralysis (*Pl.* xxiv., *fig.* 3); although in *degenerated* vessels it will be the seat of a profusion of *scavenger-cells* which entangle its walls in their processes. In the other (general paralysis), as previously stated, the morbid change is concentrated in the adventitial sheath, and is a far more acute irritative process in the loose external tunic of the vessel, which explains the more rapid implication of the nervous structures lying immediately around by direct extension. It is on this limitation of the more gross change for a time to the *inner tunic of the blood-vessels* in chronic alcoholism, that the *slow* (yet progressive) impairment of nutrition of the nerve-centres depends, which so frequently issues in steady enfeeblement of the mental faculties, akin to the advancing imbecility of senile atrophy, in which similar changes of the vessel's wall occur. It is, on the other hand, in the early implication and rapid spread of morbid activity along the *adventitial tunic of the vessels* that the more acute changes are induced in the nerve-cells of the cortex in the general paralytic. When, however, superadded to the intravascular lesions, we find sclerous tissue permeating the peripheral zone of the cortex, we have an invasion of those most externally-disposed medullated fibres which are also involved in general paralysis (*Pl.* xxiii., *fig.* 1). It is in such cases, probably, that the mental symptoms assimilate to those characteristic of general paralysis. The sclerous shrinking of the new connective formation around the extensions from the underlying ganglionic cells, results in a degeneration which is ultimately transferred to these cells themselves, inducing the already-described pigmentary and fatty degeneration preceding their absolute destruction and removal; but, this extensive atrophy of these large elements of the cortex is coincident only with the most advanced forms of alcoholic *dementia;* the earlier stage of vascular impairment, and the growth of young *scavenger-cells* in the peripheral zone, ere the cells are themselves involved, being apparently associated with the maniacal excitement and early delusional perversions of alcoholism. It is certainly a remarkable feature that in both affections we get a similar implication of the vascular channels of the pia over the almost identical motor realms of the cortex; that in both the same nervous elements are primarily involved; and that, clinically, there are presented to us so many features in common between the two affections, that it often becomes a moot point for diagnosis. This peripheral implication of the cortex would appear to us to explai the *grandiose feeling*, so frequently commingled with the delusions o persecution from which alcoholic subjects suffer; the notions of wealth of landed possessions, of exalted social status, which we find so ofte underlying delusions of restricted liberty, or of malign influenc brought to bear upon them. When, however, the motor cell an axis-cylinder process are themselves involved, then we find th characteristic delusions of persecution predominate to the exclusio often of such optimistic states; and the profound implication of th "motor element" of mind may call forth ideas of restricted volitiona freedom and reactive capacity. In all the more characteristic phase of chronic alcoholism we never fail to identify these profound lesion within the white medullated substance of the fronto-parietal lob associated with the degeneration and breaking-up of the large "*moto cells*" and *spindle-series*. Whilst, therefore, the cortical lesions general paralysis indicate an invasion from without inwards, affectin the sensory elements and apical (? sensory) poles of the motor-cell alcoholism induces, in addition thereto, extensive vascular chang from within outwards, implicating the medulla of the gyri, an effecting a destructive degeneration of the medullated fibres.

Spinal Cord.—The morbid changes found in the spinal axis ca scarcely be relegated, in any typical case of alcoholism, to one of t strict system-affections of the cord; they are too palpably of the natu of a slowly-encroaching sclerous change encircling the cord, originati in its investing membranes, and creeping inwards along the vascul tracts, and especially along the posterior median raphé. There is als undoubtedly, a tendency in such cases to a frequent implication of t posterior nerve-roots by a similarly-disposed lesion spreading into i structure from its perineural investment, inducing a change quite d similar in nature to that of the so-called multiple neuritis, which al occurs in chronic alcoholism. The investing zone of sclerosis is by means uniformly advanced at all points; more frequently we obser a decided preponderance at certain definite arcs—*e.g.*, the marginal a of the lateral column on one or on both sides; or the segment i mediately adjacent to the posterior nerve-roots; or, occasionally, section of the outer margin of the anterior root-zone; and, very f quently, the posterior median raphé, spreading thence throughout t columns of Goll. Whence this tendency to arise at different site What are the determining factors? Although we cannot reply these questions with full assurance at present, yet it is a fact of little significance that such lesions are, as in general paralysis, d tributed along the columns which are in anatomical connection wi discharging tracts at higher levels in the cerebrum; and that, as general paralysis, we find system-degenerations established apparent

Plate XXVI.


len-degenerated Nerve cell.
cular process Spider cell
Spider cell attached to the vascular wall
Degenerating Nerve cells attacked by Spider cells.
Spider cell with its vascular process
Arteriole surrounded by Spider elements

Degeneration of Nerve cells in Cortex with proliferation of the Spider or Scavenger cells Section from fifth Cortical layer in Motor region × 210.

Bale & Dani

along columns in physiological sympathy with diseased tracts higher up, yet *not by morbid continuity;* so here, also, the functional disturbances aroused in the cortex may, probably, by inducing continuous engorgement along certain spinal tracts, by the hyperactivity of their conducting strands, determine to that region the chief morbid implication. The symptoms accord with this mode of implication, for we first get decided evidence of a very chronic *lepto-meningitis*, which precedes symptoms of ascending and descending changes secondarily induced by the spread of the lesion inwards; and, still later, we find the central grey matter and special cell-groups implicated, apparently by extension along the nutrient vessels, by the same lesion. That the symptoms vary greatly in individual cases is not surprising, as they wholly depend upon *site* and *depth of implication* of the cord.

The lumbar cord may be the first affected, and the deranged sensory and motor symptoms be limited wholly to the legs; or, again, stiffness or spasm of the neck and retraction of the head may indicate cervical implication; or the dorsal region may be the site of most pronounced implications; the *vertical extent* of the meningeal affection may be slight or universal.

The constitutional state engendered in chronic alcoholic insanity is identical with what forms the basis of chronic Bright's disease; and as in this affection we have a multiplicity of local expressions of the morbid lesions, so here we find the tendency is towards a concentration in the nervous centres; atrophic states of brain, or of spinal cord, or of both combined, are thus induced from predominance of—

(*a*) Simple fatty degeneration of their nutritive vessels and tissues.
(*b*) From fatty degeneration associated with interstitial sclerosis.
(*c*) From diffuse sclerous, interstitial change.
(*d*) From periarteritis and hypertrophy of the *tunica muscularis.*

In the periarteritis, occasionally engendered in chronic alcoholics of a certain age, we probably see the pathological boundary-line overstepped betwixt simple alcoholic insanity and general paralysis of the insane; and we have resulting therefrom, in a more acute spread of the cortical lesions, what might be regarded as general paralysis accidentally evolved out of chronic alcoholism, or, as some would less correctly state the case, general paralysis *caused by alcohol.* Alcohol has its own *rôle* to play, and a most extensive one it is; but, the tissue-changes engendered thereby are always as highly characteristic as are the morbid sequences of general paralysis, and we must seek to dissever from the latter disease our notions of alcohol playing the part of a direct etiological factor, in the sense of originating the primal tissue-changes by which this disease is characterised.

In the notable thickening of the muscular tunic of the arteries seen in the spinal cord, in certain cases of alcoholic insanity, we find the general symptomatology points to the depravation of the nutrient fluids, to the especially-vitiated state of the blood, mal-assimilation, disordered digestion, deranged excretory functions, bringing in their wake the resultant changes in the arterial tunics. In such cases, as we have seen, the membranes of the cord presented no notable change, and no coarse sclerous bands of connective invaded the columns; the symptoms, which were those of an *ataxic paraplegia* of very gradual accession, were explained by the great predominance of *scavenger-cells* along the commissural end of the raphé, with morbid vascularity of the posterior columns at this site, the vessels all presenting great hypertrophy of their muscular tunic; a remarkable abundance of amyloid bodies was spread throughout the peripheral areas of the cord, and especially the posterior columns. The lateral columns exhibit a very fine punctated connective, which has induced a certain degree of atrophy of the nerve-tubuli, whilst here also the characteristic hypertrophied muscular vessels prevail abundantly throughout all regions of the cord. Nor must we fail to call attention to the fact of implication of the visceral column of the cord—the *vesicular formation* of Clarke. A very general implication of the blood-vascular system prevails; the great vessels undergo fatty and atheromatous change; and the circulatory centre itself—the heart—being an early sufferer, its muscle succumbs to fatty infiltration and degeneration, its cavities dilate, and its vital capacity is profoundly reduced. Corresponding changes appear in the large vessels at the base of the brain, which become atheromatous and distorted. It is this enfeeblement of centric circulatory energy, furthered by the retarded flow of blood in the minute vessels, which calls forth that compensatory increase of the muscular tunic of the cerebral and spinal arteries. Another factor, of great moment here for evil, must not be overlooked, that is, the diminished *vis a fronte* of cortical areas, which normally favours circulation; a failure due to the devitalisation by alcohol of the nerve-tissue, and possibly the inherited enfeeblement of neurotic descent.

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